Euphytica (2006) 147: 105–131
DOI: 10.1007/s10681-006-4600-5

Common bean breeding for resistance against biotic and abiotic stresses: From classical to MAS breeding

Phillip N. Miklas[1,*], James D. Kelly[2], Steve E. Beebe[3] & Matthew W. Blair[3]
[1]*USDA-ARS-IAREC, 24106 N. Bunn Road, Prosser, WA 99350, U.S.A.;* [2]*Crop and Soil Sciences, Michigan State University, East Lansing, MI 48824, U.S.A.;* [3]*CIAT, Apartado Aéreo 6713, Cali, Colombia*
*(*author for correspondance: e-mail: pmiklas@pars.ars.usda.gov)*

Received 6 December 2004; accepted 29 March 2005

Key words: disease resistance, drought, epistasis, gene cluster, indirect selection, insect resistance, linkage, pathogens, pests, *Phaseolus vulgaris*

Summary

Breeding for resistance to biotic and abiotic stresses of global importance in common bean is reviewed with emphasis on development and application of marker-assisted selection (MAS). The implementation and adoption of MAS in breeding for disease resistance is advanced compared to the implementation of MAS for insect and abiotic stress resistance. Highlighted examples of breeding in common bean using molecular markers reveal the role and success of MAS in gene pyramiding, rapidly deploying resistance genes via marker-assisted backcrossing, enabling simpler detection and selection of resistance genes in absence of the pathogen, and contributing to simplified breeding of complex traits by detection and indirect selection of quantitative trait loci (QTL) with major effects. The current status of MAS in breeding for resistance to angular leaf spot, anthracnose, *Bean common mosaic* and *Bean common mosaic necrosis viruses*, *Beet curly top virus*, *Bean golden yellow mosaic virus*, common bacterial blight, halo bacterial blight, rust, root rots, and white mold is reviewed in detail. Cumulative mapping of disease resistance traits has revealed new resistance gene clusters while adding to others, and reinforces the co-location of QTL conditioning resistance with specific resistance genes and defense-related genes. Breeding for resistance to insect pests is updated for bean pod weevil (Apion), bruchid seed weevils, leafhopper, thrips, bean fly, and whitefly, including the use of arcelin proteins as selectable markers for resistance to bruchid seed weevils. Breeding for resistance to abiotic stresses concentrates on drought, low soil phosphorus, and improved symbiotic nitrogen fixation. The combination of root growth and morphology traits, phosphorus uptake mechanisms, root acid exudation, and other traits in alleviating phosphorus deficiency, and identification of numerous QTL of relatively minor effect associated with each trait, reveals the complexity to be addressed in breeding for abiotic stress resistance in common bean.

Introduction

Common bean (*Phaseolus vulgaris* L.) is the most important food legume consumed worldwide. Beans provide an important source of protein (~22%), vitamins (folate), and minerals (Ca, Cu, Fe, Mg, Mn, Zn) for human diets, especially in developing countries (Broughton et al., 2003). In first-world countries the nutritional benefits and contribution of beans to healthy human diets is recognized by non-profit organizations targeting human aliments like cancer, diabetes, and heart disease (Hangen & Bennink, 2003). Annual production, including both dry and snap bean, exceeds 21 million metric tons (MT), which represents more than half of the world's total food legume production. A majority of the bean production occurs under low input agriculture on small-scale farms in developing countries. Beans produced by these resource-poor farmers are more vulnerable to attack by disease and insect pests and to abiotic stresses including drought and low soil fertility. High input farmers have more resources to combat these stresses through the use of

pesticides, fertilizers, and irrigation. Utilization of such inputs, however, can seriously reduce profitability and threaten the environment, and many pests are not effectively controlled with chemicals. Thus, across farming systems, biotic and abiotic stresses continue to represent the major constraints on subsistence production and economic yield of common bean.

Development of cultivars with improved resistance to biotic and abiotic stresses is a primary goal of bean breeding programs throughout the world. Cultivars with improved stress resistance can reduce reliance on pesticides in high input systems, avert risk of yield loss from pests in low- and high-input systems, and enable more stable bean production across diverse and adverse environments (low precipitation, high humidity, etc.) and poor soil conditions (low fertility, hillsides, etc.). This review of classical and MAS breeding for resistance to biotic and abiotic stresses in common bean will concern stresses of global importance and emphasize recent research relating to the identification, tagging, mapping, and MAS of resistance genes and quantitative trait loci (QTL). Novel and successful application of MAS will be revealed and potential disadvantages and deficiencies of MAS are also indicated.

Biotic stresses: Pathogens

Marker-assisted selection for disease resistance in common bean has been previously reviewed. Kelly and Miklas (1998) described the role of RAPDs in MAS, and extensively covered efficiency of different linkage orientations for markers linked mainly with specific resistance (SR) genes. Application of MAS in developing durable resistance through gene pyramiding, retaining defeated genes, and combining QTL of major effect were highlighted by Kelly and Miklas (1999). Miklas et al. (2002b) provided a comprehensive review of markers and linkage mapping of rust resistance genes, and Kelly and Vallejo (2004) reviewed markers, MAS, map location, and breeding value of major anthracnose genes. The recent comprehensive map of disease resistance traits in common bean reveals numerous resistance gene clusters (Kelly et al., 2003), including co-location of genes for resistance to anthracnose and rust. Co-location of disease resistance QTL with putative candidate SR gene clusters and defense-related genes (Geffroy et al., 2000) is becoming more visible in the genome (Figure 1). Resistance gene clusters possessing genes known to derive from the same gene pool (Andean versus Middle American origin; Singh et al., 1991; Beebe et al., 2000) have been discovered (Kelly et al., 2003), and reinforce the duplication and divergence of genes from ancestral resistance genes and gene clusters (Geffroy et al., 1999), and supports previous findings of co-evolution of host resistance and pathogen virulence diversity at the gene pool level which is worthy of further explanation herein.

Co-evolution of host and pathogen has lead to isolates (pathotypes) of Andean origin which attack beans primarily from the Andean gene pool. Conversely, isolates of Middle American origin attack beans primarily in the Middle American gene pool but possess a wider range of virulence also infecting beans of Andean origin. Similar co-evolution of pathogen virulence with common bean gene pools has been observed for the angular leaf spot (Guzmán et al., 1995; Pastor-Corrales & Jara, 1995), anthracnose (Balardin & Kelly, 1998; Islam et al., 2002), common bacterial blight (Mkandawire et al., 2004) and rust pathogens (Sandlin et al., 1999). Co-evolution of pathogen virulence within gene pools affects resistance gene deployment strategies. Resistance genes of Middle American origin are very effective when transferred to beans of Andean background and deployed in regions where Andean isolates prevail (East Africa, Colombia, Ecuador). Similarly, genes of Andean origin are very effective when transferred to beans of Middle American background and deployed in regions where isolates of Middle American origin prevail (Central America, Mexico, USA). The development of lines with resistance genes from both gene pools, detailed in anthracnose and rust sections below, is a recognized strategy for developing improved, broad-based, resistance in bean.

Given that many disease resistance genes in common bean exist in gene clusters at complex loci (Kelly et al., 2003), it is becoming increasingly important to understand the physical arrangement and sequence diversity of disease resistance gene families in the crop. Two candidate gene tagging approaches have become important for analyzing resistance genes: these include the cloning of resistance gene analogs (RGAs) (Rivkin et al., 1999; Vallad et al., 2001; Ferrier-Cana et al., 2003; López et al., 2003) and development of targeted region amplified polymorphisms (TRAP) (Hu and Vick, 2003). Both techniques generate molecular markers that could be useful for selection of resistance genes or for dissecting resistance gene clusters. For common bean, the integration of MAS with classical approaches in breeding for disease resistance is advancing rapidly as described later for specific bacterial, fungal, and viral diseases of global importance.

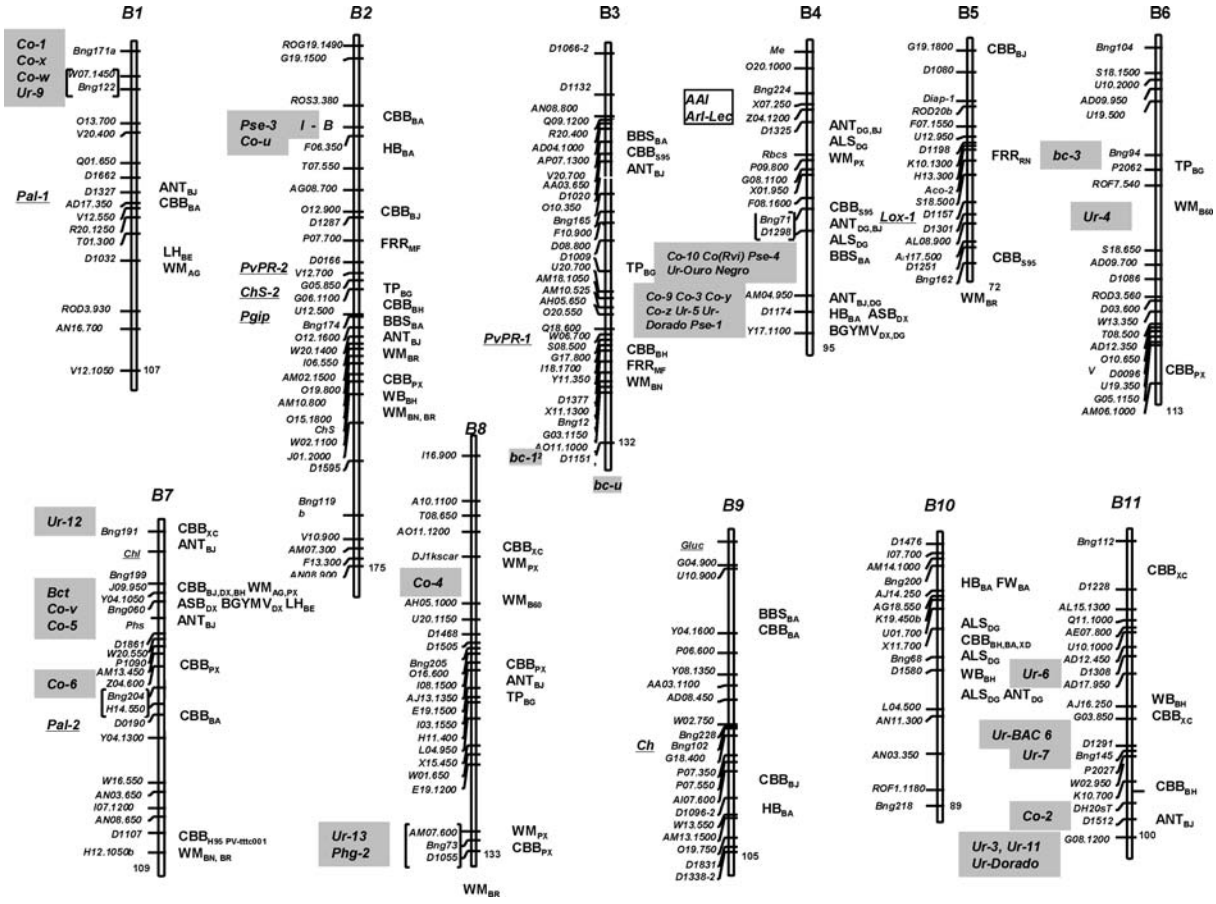

Figure 1. Comprehensive genomic map of disease and insect resistance genes and QTL in common bean. The linkage groups correspond to the core map version of Freyre et al. (1998), and resemble the maps presented by Kelly et al. (2003), and Kelly and Vallejo (2004). Directly to the left of each linkage group are the framework molecular markers (smaller font), the monogenic disease resistance genes (shaded boxes), defense-related genes (underlined), and arcelin, lectin and alpha-amylase inhibitor genes (clear box). The *Co* are anthracnose resistance loci, *Ur* rust resistance loci (*Ur-Dorado*, *Ur-Ouro Negro*, and *Ur-BAC 6* refer to the line source of unnamed genes), *Pse* halo blight resistance loci, *I* and *bc* are dominant and recessive genes respectively for resistance to BCMV, *Phg* angular leaf spot resistance locus, and *Bct* is a locus for resistance to BCTV. For further explanations on DNA markers and gene symbols see Gepts (1999) and Bassett (2004). To the right of each linkage group are QTL mapped in different populations. ALS: resistance to angular leaf spot, ANT: anthracnose, ASB: ashy stem blight, BGYMV: bean golden yellow mosaic virus, BBS: bacterial brown spot, CBB: common bacterial blight, FRR: *Fusarium* root rot, HB: halo blight, LH: leafhopper, TP: thrips, WB: web blight, and WM: white mold resistance. Symbols in subscript represent the source population of the QTL. AG: A55/G 122 (Miklas et al., 2001), BA: Belneb-RR-1/A55 (Ariyarathne et al., 1999; Fourie et al., 2004; Jung et al., 2003), BE: Berna/EMP 419 (Murray et al., 2004a,b), BG: BAT 881/G 21212 (Frei et al., 2005), BJ: BAT 93/Jalo EEP558 (Freyre et al., 1998; Gepts, 1999; Geffroy et al., 2000), BH: BAC 6/HT 7719 (Jung et al., 1996), BN: Bunsi/Newport (Kolkman & Kelly, 2003), BR: Bunsi/Raven (Ender & Kelly, 2005), B60: Benton/NY6020-4 (Miklas et al., 2003b), DG: DOR 364/G 19833 (López et al., 2003), DX: DOR 364/XAN 176 (Miklas et al., 2000c), H95: HR67/OAC 95 (Yu et al., 2004), MF: Montcalm/FR266 (Schneider et al., 2001), PX: PC50/XAN 159 (Jung et al., 1997, 1998; Park et al., 2001), RN: Red Hawk/Negro San Luis (Román-Avilés & Kelly, 2005), S95: Seaforth/OAC 95 (Tar'an et al., 2001), and XC: XR-235-1-1/Calima (Yu et al., 1998). Gene and QTL locations are approximate because most were not directly mapped in the BAT 93/Jalo EEP558 population. The total distance of each linkage group is expressed in Kosambi cM (bottom-right).

Angular leaf spot

Angular leaf spot, caused by the fungal pathogen *Phaeoisariopsis griseola* (Sacc.) Ferraris, is a serious disease in tropical and sub-tropical countries of South America, Central America, and East Africa. Angular leaf spot is rated the most important and widespread biotic constraint afflicting bean production in Africa (Wortmann et al., 1998). An integrated control strategy employing use of pathogen-free seed, cultural practices, and fungicides is useful, but genetic resistance provides better and more economical

control. Genetic resistance is mostly monogenic and race-specific, but because the pathogen is highly variable with many different races characterized (Busogoro et al., 1999b; Mahuku et al., 2002), combinations of genes from diverse sources are needed to provide broad resistance to an array of races prevalent in a region. Pyramiding genes with specificities for resistance against the same races(s) predominant in a region is a breeding strategy used to improve the durability of major genes that combat hypervariable pathogens. Diversity studies with this pathogen were the first to reveal co-evolution of angular leaf spot pathogen with the gene pools of the common bean host (Guzmán et al., 1995; Pastor-Corrales & Jara, 1995).

Initial screening of the common bean collection (~20,000 accessions) at CIAT (the International Center of Tropical Agriculture in Cali, Colombia) uncovered few sources of resistance to angular leaf spot (Schwartz et al., 1982). Recently, expanded evaluations reveal the secondary gene pool (*P. coccineus* and *P. polyanthus*) as an abundant source of resistance (Busogoro et al., 1999a; Mahuku et al., 2003). Mahuku et al. (2003) identified 78 interspecific dry bean lines with resistance putatively transferred from the secondary gene pool, which represents important germplasm for future utilization. Traditional breeding at CIAT involving hybridization among resistance sources in single or multiple interracial crosses followed by selection under disease pressure in field nurseries and greenhouse screening trials has resulted in development of germplasm lines MAR 1, MAR 2, MAR 3, AND 277, and CAL 143 with improved broad-based resistance to angular leaf spot (Aggarwal et al., 2004; Singh et al., 2003). Other important sources of resistance include BAT 332, MEX 54, Cornell 49–242, Ouro Negro, G 10474, and other *P. vulgaris* landrace accessions and bred lines listed by Pastor-Corrales et al. (1998), Mahuku et al. (2003), and Beebe and Pastor-Corrales (1991).

Inheritance studies reveal that resistance present in BAT 332 (Caixeta et al., 2003), Mexico 54 (Sartorato et al., 2000), Cornell 49-242 (Nietsche et al., 2000), Ouro Negro (Corrêa et al., 2001), AND 277 (Carvalho et al., 1998), MAR 2 (Ferreira et al., 2000), and G 10474 (Mahuku et al., 2004) is conditioned primarily by single dominant genes. Monogenic resistance genes with recessive inheritance have also been reported (Corrêa et al., 2001; Santos-Filho et al., 1976). RAPD or SCAR markers linked with many of the dominant resistance genes have been obtained (see SCAR list, Miklas, 2005). The SN02 SCAR marker linked with *Phg-2* gene was identified in Mexico 54 (Sartorato et al., 2000) and cosegregated with a dominant resistance gene in Cornell 49-242 (Nietsche et al., 2000). Both lines are in the host differential series (Pastor-Corrales et al., 1998), but Cornell 49-242 with a binary code rating of 32 is more resistant than Mexico 54 with a rating of 8. Cornell 49-242 must either have additional genes for resistance or possess a more effective allele at the *Phg-2* locus.

Five QTL for angular leaf spot resistance were identified in the DOR 364 × G 19833 population and mapped to linkage groups B4 and B10 (Figure 1) (López et al., 2003). All five QTL were located near RGAs suggesting that they share structural similarities with R genes, and perhaps reside within gene clusters because resistance to anthracnose co-located with three of the QTL. The utility of these QTL for breeding purposes has not been fully explored.

The SN02 SCAR marker linked with *Phg-2* (Sartorato et al., 2000), in our laboratory (PNM), mapped directly in the BAT 93 × Jalo EEP558 population to a terminal end of linkage group B8 (Figure 1). The map locations for the other tagged angular leaf spot resistance genes are unknown. Although, *Phg-2* is reported to be independent from *Phg-1* identified in AND 277 (Carvalho et al., 1998; Queiroz et al., 2004), definitive allelism tests of independence have not been published. AND 277 and the derived line CAL 143 represent an important breakthrough as the first Andean beans with useful levels of resistance to angular leaf spot (Aggarwal et al., 2004). The linked markers (Miklas, 2005) for *Phg-1*, *Phg-2*, and for the genes from G 10474, Ouro Negro, BAT 332 (RAPD AA07.950) and MAR 2 (RAPD E04.500) enable MAS of resistance derived from diverse sources, and indeed MAS for resistance to ALS is being conducted in Brazil (Oliveira et al., 2002; Ragagnin et al., 2003). Utilization of the markers for pyramiding resistance genes is hampered, however, by a lack of knowledge about genomic distribution of the genes. In the absence of allelism tests, locating the markers on the linkage map would help to determine gene independence, and relationship or lack thereof with QTL for angular leaf spot resistance (López et al., 2003).

For East Africa MAS is being used to develop backcross lines with resistance to diseases caused primarily by hypervariable pathogens such as angular leaf spot, anthracnose, and rust. East African bean varieties are being used as the recurrent parents to maintain local genetic diversity. Local variety mixtures in East Africa will be supplemented with the backcross resistant lines

to provide stable disease control. Supplementing local mixtures with resistant lines has been shown to provide protection against angular leaf spot in Zaire (Pyndji & Trutmann, 1992).

Anthracnose

Bean anthracnose, caused by *Colletotrichum lindemuthianum*, is a highly variable seed-borne fungal pathogen of common bean that is found on all continents where beans are grown (Melotto et al., 2000). Resistance to anthracnose is conditioned primarily by nine major independent genes *Co-1* to *Co-10*, as Méndez-Vigo et al. (2005) recently showed that the *Co-3* and *Co-9* genes are allelic. With the exception of the recessive *Co-8* gene, all other nine are dominant genes and multiple alleles exist at the *Co-1*, *Co-3* and *Co-4* loci (reviewed by Kelly & Vallejo, 2004). The nine resistance genes *Co-2* to *Co-10* are Middle American in origin and *Co-1* is the only locus from the Andean gene pool. An order of dominance exists among the four alleles at the *Co-1* locus.

Eight resistance loci have been mapped (Figure 1) to the integrated bean linkage map (Freyre et al., 1998) and the three *Co*-genes that map to linkage groups B1, B4 and B11 cluster with the *Ur*-genes for rust resistance (Kelly et al., 2003; Miklas et al., 2002b). The *Co-1* gene resides on B1; *Co-2* on B11, *Co-3/Co-9* on B4 (Méndez-Vigo et al., 2005); *Co-4* on B8 (Melotto et al., 2004); *Co-5* (Campa et al., 2005) *Co-6* on B7; and *Co-10* on B4 (Kelly & Vallejo, 2004). With the exception of the *Co-3*/*Co-9* gene cluster on B4, none of the other major *Co*-genes appear to be linked. In addition, there is co-localization with major resistance genes and QTL that condition partial resistance to anthracnose (Geffroy et al., 2000). The 10 *Co*-genes are represented in the anthracnose differential cultivars (Melotto et al., 2000), but are present as part of a multi-allelic series or in combination with other *Co*-genes, making the characterization of more complex races of *C. lindemuthianum* difficult. Although the *Co*-genes behave as major Mendelian factors, they most likely exist as resistance gene clusters as has been demonstrated at the molecular level for the B4 R-gene cluster (Ferrier-Cana et al., 2003).

Molecular markers linked to the majority of major *Co*-genes have been widely reported and these provide the opportunity to enhance disease resistance through MAS (reviewed by Kelly & Vallejo, 2004; Kelly et al., 2003). Pyramiding genetically diverse resistance genes using MAS and deploying different gene combinations in different geographic regions is proposed as the most practical and realistic approach to provide efficient long-term control of bean anthracnose (Balardin & Kelly, 1998). MAS has been used successfully to breed for enhanced resistance to anthracnose in the cultivar Perola in Brazil (Ragagnin et al., 2003) and in pinto beans in the United States (Miklas et al., 2003c), but there is a need for caution based on the unsuccessful attempts to introgress the *Co-4*^{2} gene using marker-assisted backcrossing in two landrace bean cultivars from Ecuador (Ernest & Kelly, 2004). Indirect selection should be periodically verified by direct selection to ensure that the resistance gene is being transferred.

Bean breeders have a unique opportunity to improve on natural gene pyramids in landrace cultivars (Young et al., 1998) by combining resistance genes from the two major gene pools to develop complementary resistance to a wide range of pathogenic races. To design effective gene pyramids, breeders need information on pathogenic variability of *C. lindemuthianum* present in production areas. For example, 16 races of *C. lindemuthianum* from Guatemala (Muhuka, personal communication, 2004) defeated the *Co-2*, *Co-5*, *Co-6* genes and supported the potential value of the *Co-1* and *Co-4* genes both of which have suffered major breakdown of resistance in Ecuador and Mexico, respectively. In North America, combining the *Co-4*^{2}, *Co-5* and *Co-6* genes would be effective, whereas for areas of Central America the most suitable gene pair would be the *Co-1*^{2} and *Co-4*^{2} gene combination. Since the *Co-4*^{2} is recognized as the most broadly-based resistance gene (Balardin & Kelly, 1998), it would be invaluable to include in gene pyramids with other Middle American genes in those countries like the Dominican Republic and Ecuador where Andean races prevail. Since the genes differ in their effectiveness in controlling the highly variable races of the anthracnose pathogen, continued evaluation of resistance sources suggests that better tailored gene pyramids can be developed provided information is available on the race diversity in specific regions. When selecting for anthracnose resistance in a particular region, bean breeders should carefully choose a gene pair that, if deployed singly, would confer resistance to all known races in that region.

Bean common mosaic virus (BCMV) and bean common mosaic necrosis virus (BCMNV)

Bean common mosaic virus and BCMNV are the most common and destructive potyviruses known to infect

common bean worldwide (Drijfhout, 1978; McKern et al., 1992). Both viruses are seed-borne and transmitted by several aphid species in a non-persistent manner (Drijfhout, 1978). Necrotic strains evolved more recently in the African continent (Spence & Walkey, 1995) as recombination between strains of BCMV and BCMNV has been reported (Larsen et al., 2005). Genetic resistance to both potyviruses is conditioned by a series of independent multi-allelic loci in common bean (Drijfhout, 1978). The dominant *I* gene that confers hypersensitive resistance to five related potyviruses (BCMV, BlCMV, CAMV, SMV and WMV; Kyle & Provvidenti, 1993), has also been the focus of positional gene cloning activities (Vallejos et al., 2000). The *I* gene located on B2 (Kelly et al., 2003), is independent of recessive resistance conditioned by three different *bc* genes. The *bc-3* gene is located on B6 (Johnson et al., 1997; Miklas et al., 2000c; Mukeshimana et al., 2005), whereas the *bc-1*^{2} allele was mapped to B3 (Miklas et al., 2000a). The non-specific *bc-u* allele, needed for expression of *bc-2*^{2} resistance, also resides on B3 based on the loose linkage with the *bc-1* locus (Strausbaugh et al., 1999).

The independence of the BCMV resistance genes provides opportunities to use gene pyramiding as a strategy in breeding for durable resistance. Bean breeders recognize that the combination of the dominant *I* gene with recessive *bc* resistance genes offers durability over single gene resistance to BCMV and BCMNV, since the two types of genes have distinctly different mechanisms of resistance (Kelly, 1997). The dominant *I* gene is defeated by all necrotic strains, whereas the three most effective recessive genes (*bc-1*^{2}, *bc-2*^{2}, *bc-3*) act constitutively by restricting virus movement within the plant, probably through the virus movement proteins. The action of the dominant *I* gene is masked by the recessive *bc-3* gene, so as efforts to incorporate the *bc-3* gene into new germplasm proceed, the risk of losing the *I* gene in improved germplasm increases, since direct selection for the *I* gene is not possible. Linked markers offer the only realistic opportunity to maintain and continue to utilize the *I* gene as a pyramided resistance gene in future bean cultivars. A marker tightly linked to the *I* gene (Haley et al., 1994b; Melotto et al., 1996) has been demonstrated in many laboratories to be effective across a wide range of germplasm from both gene pools. Breeders (Kelly et al., 1994; Miklas et al., 2002a; Miklas & Kelly, 2002) have used markers linked to the *I* gene to develop enhanced germplasm with the *I* + *bc-3* gene combination. A linkage distance of ~5 cM between the *I* gene and linked SW13 marker may result in recombinants that possess the marker but lack the gene (Vandemark & Miklas, 2005), so pathogen testing a final testcross is recommended to confirm presence of the *I* gene.

Markers linked in repulsion to the *bc-3* gene have been identified (Haley et al., 1994a; Johnson et al., 1997), but direct screening with strains of BCMNV is still required to confirm the presence of the *bc-3* gene. The AD19 marker linked in coupling (Haley et al., 1994a) with *bc-3* was ineffective for MAS of *bc-3* in susceptible germplasm of Mesoamerican origin because the marker was ubiquitous in this gene pool. Miklas et al. (1996a) described recombinant-facilitated MAS as a means to overcome the gene-pool specificity of resistance-linked markers. For example, this method requires identifying a recombinant individual in a segregating population that possesses the gene (*bc-3*) but not the linked marker (AD19). The recombinant line is crossed with susceptible Middle American lines that possess the marker (AD19) followed by MAS against the marker in the resulting segregating population to retain progeny with *bc-3* resistance. Repulsion markers, however, are not conducive to rapid deployment of genes via backcrossing because they cannot distinguish F_1 that possess the linked gene. Codominant markers are able to distinguish F_1 but generally are unavailable for MAS in common bean. Recently, Vandemark and Miklas (2002, 2005) described codominant interpretation of dominant markers using quantitative PCR to enable discrimination of homozygous and heterozygous individuals for *I* and *bc-1*^{2} genes. The efficiency of MAS is greatly enhanced with codominant markers, codominantly interpreted dominant markers, or linked marker pairs in coupling and repulsion orientation with the target gene (Haley et al., 1994a; Johnson et al., 1995; Kelly & Miklas, 1998; Vandemark & Miklas, 2002).

New opportunities exist to improve virus resistance using MAS for a tightly linked (3.5 cM) codominant AFLP marker, $E_{ACA}M_{CGG}$ 169/172 that with the OG6.595 RAPD marker flanked the *bc-3* gene (Mukeshimana et al., 2005). Caution must be used when deploying the *bc-3* gene singly because some lines with putative *bc-3* resistance have been observed to be susceptible to several common strains of BCMV (Larsen et al., 2005). Clearly gene pyramiding is a workable strategy in breeding beans for virus resistance as the various resistance genes that reside on different linkage groups provide contrasting modes of resistance patterns to the diverse strains of BCMV and BCMNV (Kelly et al., 1995, 2003).

Beet curly top virus (BCTV)

Beet curly top virus is a geminivirus disease of common bean vectored by the beet leafhopper *Circulifer tenellus* (Baker). The virus is endemic to the semiarid regions of the western United States, but occurs worldwide (ICTVdB database), and infects 300 other plant species including sugar beet (*Beta vulgaris* L.), tomato (*Lycopersicon esculentum* Mill.), and pepper (*Capsicum frutescens* L.) (Bennett, 1971). Genetic resistance provides the most effective control of BCTV in bean. Although numerous cultivars with effective levels of resistance have been developed (Sutton & Coyne, 2002), breeding for resistance is difficult because field epidemics are sporadic and non-uniform, and greenhouse evaluations require either viruliferous leafhoppers or *Agrobacterium tumefaciens*-mediated infectious clones to infect bean plants (Elmer et al., 1988; Stenger et al., 1991).

Schultz and Dean (1947) described a dominant and recessive digenic model of inheritance for resistance to BCTV. Larsen and Miklas (2004) generated a SCAR marker linked with a dominant resistance gene *Bct* believed to be the same gene identified over 50 years ago (Schultz & Dean, 1947). The *Bct* gene conditions a high level of resistance to BCTV. Given the difficulty of conducting field and greenhouse evaluations, MAS for *Bct*, using the SAS8.1550 SCAR marker, is quickly being adapted by bean breeders for developing BCTV-resistant cultivars in the absence of the pathogen. It is noteworthy that SAS8.1550 is only useful for MAS in beans of Andean origin because of its ubiquitous presence in beans of Middle American origin including those susceptible to BCTV. MAS restricted to specific gene pools (see the section on BCMV in this paper) is a common occurrence for resistance-linked markers in bean (reviewed by Kelly & Miklas, 1999; Miklas et al., 1993, 1996a).

The *Bct* gene is located on linkage group B7 (Larsen & Miklas, 2004) in the vicinity of other genes/QTL that condition resistance to anthracnose, *Bean golden yellow mosaic virus* (BGYMV), common bacterial blight (CBB), Macrophomina, and white mold, suggesting that the gene may be a component of a resistance gene cluster. There is interest in the potential of *Bct* as a transgene to combat BCTV in other crops like pepper and tomato, and studies are underway in our laboratory to determine the cross resistance of *Bct* against other geminiviruses that infect bean including *Bean dwarf mosaic virus* (BDMV), *Bean calico mosaic virus* (BCaMV), and BGYMV.

Bean golden yellow mosaic virus (BGYMV)

Bean golden yellow mosaic virus is a whitefly-transmitted geminivirus disease that occurs in the tropics and sub tropics of Latin America (Gálvez & Morales, 1989). Genetic resistance is the most critical component of integrated strategies used to control the disease in commercial bean production fields. CIAT breeders working with national program scientists in Guatemala and Honduras used phenotypic recurrent selection to develop cultivars with combined sources of resistance from the Mesoamerican and Durango races (Beebe, 1994; Beebe & Pastor-Corrales, 1991). Similarly, inter-gene pool crosses (Singh et al., 2000a) were used to combine resistance from the Middle American and Andean gene pools to attain high levels of resistance, as in breeding lines GMR-1 and GMR-5 (Singh et al., 2000b). The Andean dark red kidney bean Royal Red seems to be an important contributor to the resistance present in the GMR lines. Subsequently, developed cultivars for the Caribbean and Central America combine BGYMV resistance across races and gene pools (Beaver et al., 2003). The most recent breeding advance has been interspecific crosses that have yielded adapted lines (Beaver et al., 2005) with novel resistance to BGYMV derived from *P. coccineus*, a well-known but underutilized source of resistance from the secondary gene pool (Beebe & Pastor-Corrales, 1991; Bianchini et al., 1994).

Inheritance studies involving many of the sources mentioned earlier reveal that both major genes (*bgm-1*, *bgm-2*, *Bgp-1*) and QTL condition resistance (see review Kelly et al., 2003). The dominant gene *Bgp-1* found in Don Silvio conditions normal pod development under severe disease pressure, but appears to require the presence of *bgm-1* for expression (Acevedo-Román et al., 2004). A recessive gene conditioning resistance to chlorosis and a dominant gene conditioning normal pod development were identified in a breeding line with resistance derived from *P. coccineus* accession G 35172 (Osorno et al., 2003). Allelism tests indicate the two genes are independent of *bgm-1*, *bgm-2*, and *Bgp-1*. Other researchers reported similar two-gene inheritance for BGYMV resistance in *P. vulgaris* × *P. coccineus* interspecific populations (Bianchini et al., 1994).

The two major QTL identified by Miklas et al. (1996b) that condition reduced mosaic reside within clusters of genes, e.g. *Co-9*, *Ur-5*, and *Pse-1* on B4, and among major genes and QTL conditioning resistance to BCTV (also a geminivirus), CBB, white mold,

anthracnose, and *Macrophomina* (ashy stem blight) on B7 (Figure 1). The primers for the SW12 SCAR (Miklas et al., 2000c; Singh et al., 2000a) linked with the BGYMV QTL on B4 amplified different size fragments that cosegregated codominantly (2.2 cM) with the *Co-9* gene (Méndez-Vigo et al., 2005), which further supports presence of the BGYMV-resistance QTL within the B4 resistance gene cluster. López et al. (2003) also identified a RGA that was linked to this same QTL on B4. The B4 QTL linked with SW12.700 SCAR and *bgm-1* gene linked with the codominant SR2 SCAR marker (Urrea et al., 1996) are the only genes (to date) for resistance to BGYMV amenable to MAS. At CIAT, MAS for resistance genes for BGYMV is now practiced routinely. The MAS system has improved in efficiency over several years whereby 3000 plants were originally evaluated for one SCAR (SR2) marker for *bgm-1* in 57 person-days, and eventually this was reduced to 26 person-days. Plants are tagged and numbered individually in the field, young leaves are sampled directly into titer plates, and alkaline extraction is practiced. Recently, a protocol has been developed to multiplex the amplification of the *bgm-1* SCAR (SR2) with a SCAR (SW12) for the QTL on B4, greatly increasing the efficiency of MAS. As many as 20,000 reactions are run in a year.

In summary, high levels of resistance to BGYMV are obtained by combining resistance sources from diverse backgrounds. For example, Don Silvio, which is highly resistant, possesses three resistance genes from different sources and with different functions: *bgm-1* conditioning non-chlorosis derived from Garrapato via breeding line A429, *Bgp-1* conditioning normal pods probably derived from BAT 1215, and SW12 linked QTL on B4 conditioning delayed and reduced mosaic symptoms derived from Porrillo Sintetico. MAS will play an ever-increasing role in breeding for BGYMV resistance because disease screening in the field is unpredictable and greenhouse screening is inefficient. The pole garden bean cultivar Genuine (Shamrock Seed Co., Salinas, CA), with moderate resistance to BGYMV, was developed using MAS for the *bgm-1* marker (Stavely et al., 2001). Markers are needed for *bgm-2*, *Bgp-1*, and the newly identified genes from *P. coccineus* to facilitate utilization of these resistance genes by breeders.

Common bacterial blight

Common bacterial blight (CBB) caused by *Xanthomonas axonopodis* pv. *phaseoli* is a seed-borne disease that plagues bean production worldwide. Clean seed programs, chemicals, cultural practices, and genetic resistance are used to control this disease (Coyne et al., 2003). Breeding for genetic resistance is complex as revealed by identification of 22 QTL distributed across all 11 chromosomes (Figure 1). Expression of these QTL is influenced by environment, disease pressure, plant maturity and plant organ: seed, leaf, and pod (Ariyarathne et al., 1999; Jung et al., 1997; Miklas et al., 1996b; Santos et al., 2003; reviewed by Kelly & Miklas, 1999, and Kelly et al., 2003).

SCAR markers BC420, SU91, and SAP6 linked with three major QTL on B6, B8, and B10 (see review by Kelly et al., 2003), respectively, are being used for MAS of CBB resistance (Mutlu et al., 2005a; Yu et al., 2000) and to validate QTL present in resistant lines selected by phenotypic selection (Fourie & Herselman, 2002). In fact, all three markers can be multiplexed in a single PCR reaction (Miklas et al., 2000b) to expedite MAS for combined resistance to CBB (Figure 2). Thus far, of the three SCAR markers, SU91 linked with the

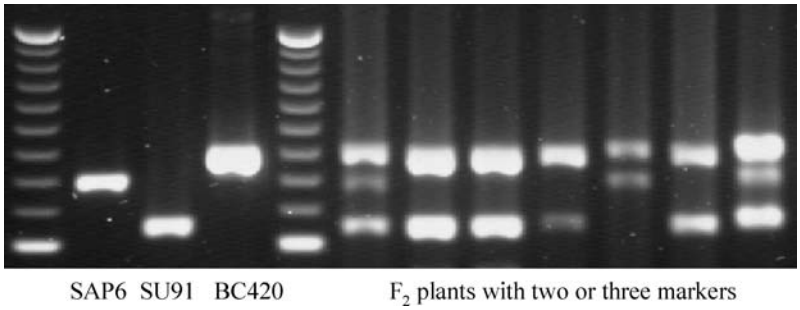

Figure 2. Agarose gel photograph depicting SCAR markers linked with QTL on putative linkage groups B10 (SAP6, lane 2), B8 (SU91, lane 3), B6 (BC420, lane 4), and multiplexed in single PCR reactions for DNA from F2 plants segregating for all three SCAR markers (lanes 6–12). Lanes 1 and 5 represent 100 bp ladder starting at 500 bp.

major QTL tentatively located on B8 has been utilized the most for MAS (Miklas et al., 2005b; Mutlu et al., 2005b). It is important to note that breeding strategies that combine MAS with intermittent phenotypic selection have been the most effective in developing lines with improved CBB resistance. Phenotypic selection is needed to retain minor effect QTL and select for epistatic interactions that contribute to improved resistance.

The BC420 and SU91 SCAR markers and respective linked QTL derive from tepary bean (Miklas et al., 2000b) via breeding line XAN 159 (Thomas & Waines, 1984). Recently, Yu et al. (2004) mapped BC420 marker and a linked SSR marker to the end of linkage group B7, so there is some discrepancy as to the location of this QTL. Translocation could explain the movement of traits from the end of one linkage group B6 to the end of another B7. Location of BC420 to linkage group B6 was based primarily on linkage with *V* locus that conditions purple flower color and dark seed colors (Jung et al., 1997). The integration of *V* may be misplaced or the gene influencing seed color linked with BC420 may also derive from tepary bean and occur at a different locus than *V*. Nonetheless, repeated attempts to recover the resistance QTL linked with BC420 marker in seed types other than white, black, or black-mottled has failed. White seed color beans possess the recessive *p* gene, which is epistatic to all other seed coat color genes. Thus, white-seeded beans with BC420 QTL like HR67 (Yu et al., 2004) and CBB-resistant-Teebus (Fourie & Herselman, 2002) can possess *V* or other seed coat color genes because the genes are not expressed.

A recent study (Miklas et al., 2003a) showed that the great northern landrace cultivar Montana No. 5 was the source of the QTL (linked with SAP6 and putatively located on B10) in Great Northern Nebraska No. 1 Sel. 27 (GN No. 1 Sel. 27), not tepary bean as originally thought. Other markers linked with resistance traits in bean have been used to trace resistance back to its source (e.g. SR2 marker revealed Garrapatos as the source of *bgm-1*). Most cultivars around the world bred with CBB resistance possess the SAP6 marker, and have GN No. 1 Sel. 27 or a GN No. 1 Sel. 27 derived line in their pedigree.

Two other major QTL have been identified, on B5 and B7 (Figure 1), but MAS for them has not yet been developed. A major QTL present in OAC 95-4 (OAC REX) maps toward the end of linkage group B5 (Tar'an et al., 2001). A QTL located on B7 near the *Phs* locus derives from either GN No. 1 Sel. 27 or PI 207262 and has been identified in three independent studies (Jung et al., 1996; Miklas et al, 1996b; Nodari et al., 1993).

Bean breeders, using traditional breeding approaches, have combined resistance sources from the primary and secondary gene pools to obtain cultivars and lines with improved resistance to CBB (Table 1). The VAX lines with combined resistance from *P. vulgaris* and *P. acutifolius* possess the highest level of CBB resistance developed to date (Singh et al., 2001). Higher levels of resistance coincide with an increase in the number of sources combined, and is

Table 1. Representative dry bean lines and cultivars with resistance to common bacterial blight (CBB) derived from individual or combined sources

	Sources				
	P. vulgaris				
Lines & cultivars	Montana No. 5	PI 207262	*P. coccineus*	*P. acutifolius* (Tepary markers)	Disease score (1–9)[a]
Jules, Chase, Montcalm	×				6, 7, 8
XAN 112, BAT 93	×	×			4, 5, 6
XR 235-1-1			×		5, 6
USPT-CBB-1	×		×		4, 5, 6
OAC 88-1				× (SU91)	4, 5, 6
HR 67				× (BC420)	3, 4, 5
XAN 159, CBB-Teebus				× (SU91 and BC420)	2, 3, 4
ABCP-8, USDK-CBB-15	×			× (SU91)	3, 4, 5
Wilkinson 2	×		×	× (SU91 and BC420)	2, 3, 4
XAN 309, VAX lines 3–6	×	×		× (SU91)	1, 2

[a]Relative average disease scores (1–9, where 1 is no visible symptoms and 9 is completely diseased) compiled from published and unpublished data.

also dependent on source, with *P. acutifolius* derived resistance exhibiting the highest level, followed by *P. coccineus*, then *P. vulgaris* (Singh & Muñoz, 1999). Now breeders have MAS available to facilitate accumulation of QTL from diverse sources described earlier to attain high levels of CBB resistance in new bean cultivars. The markers also provide tools for investigating genetic interactions among the resistance QTL, which may lead to improved resistance gene deployment strategies in the future.

Halo blight

Halo blight is a seed-borne bacterial disease (caused by *Pseudomonas syringae* pv. *phaseolicola*) (*Psp*) that limits common bean production across humid and cool climatic zones worldwide. Genetic resistance is the most effective control method. A host/pathogen differential series developed by Taylor et al. (1996a,b) identifies five monogenic resistance genes, none of which condition resistance to Race 6, the most prevalent race of the pathogen in bean production regions of East Africa and the United States (Lamppa et al., 2002; Taylor et al., 1996a). Prevalence of Race 6 necessitates incorporating quantitative resistance from sources such as CAL 143, GN No. 1 Sel. 27, and PI 150414, which provide effective broad-based resistance against all races of the pathogen. Efforts are underway to tag and map the resistance QTL from these sources. Resistance from PI 150414 is already widely dispersed in snap bean germplasm (Silbernagel & Hannan, 1992). Currently, MAS for halo blight resistance is not being conducted due to a lack of resistance-linked markers.

Quantitative trait loci for resistance to halo blight were identified in BelNeb-RR-1/A 55 RIL population (Ariyaranthne et al., 1999). Using the same population, Fourie et al. (2004) observed that three of the QTL corresponded with the location of *Pse-1*, *Pse-3*, and *Pse-4* genes on linkage group B4, B2, and B4, respectively (Figure 1). The *Pse-1* gene, which conditions resistance to Races 1, 7, and 9, resides within the B4 cluster of genes and QTL conditioning anthracnose, rust, ashy stem blight, BGYMV and bacterial brown spot (caused by *P. syringae* pv. *syringae*) resistance. QTL and genes with monogenic inheritance for resistance to halo blight have been observed within the same gene cluster similar to observations with anthracnose (Geffroy et al., 2000). Sequence data of the SB10.520 SCAR marker, tightly linked with *Pse-1*, closely aligns with DNA sequence of a RGA associated with anthracnose resistance and mapped in the same region of B4 (unpublished data).

The *Pse-3* gene which conditions resistance to Races 3 and 4 is tightly linked with the *I* gene, as no recombinants for these two genes have been observed (Taylor et al., 1996b). Both genes condition a hypersensitive reaction, *Pse-3* to *Psp* Races 3 and 4 and *I* gene to all strains of BCMNV and certain strains of BCMV expressing temperature-sensitive necrosis. Given a similar hypersensitive mode of action for both *Pse-3* and *I* and the lack of recombination between genes, it is possible that *I* gene is conditioning resistance to both diseases. The *Pse-4* gene that conditions resistance to Race 5 is loosely linked with *Pse-1* (Figure 1) which may explain why both genes are present in the differential dry bean cultivar UI-3. In an ongoing study (PNM), allelism tests between BelNeb-RR-1 and UI-3 indicates they may possess different genes (∼*Pse-1*) that condition resistance to Races 1, 7, and 9.

Root rot

Root rot, caused by a complex of soil-borne pathogens that include: *Fusarium solani* f. sp. *phaseoli,* Fusarium root rot; *Fusarium oxysporum* f. sp. *phaseoli,* Fusarium wilt or yellows; *Rhizoctonia solani,* Rhizotonia root rot; *Pythium* spp., Pythium wilt and seed rot; *Macrophomina phaseolina*, charcoal rot or ashy stem blight; *Thielaviopsis basicola*, black root rot; and *Aphanomyces eufeches* f. sp. *phaseoli,* Aphanomyces root rot, is a major limiting disease of common bean (Abawi & Pastor-Corrales, 1990). Root rots are economically important in most bean production areas (Snapp et al., 2003) but are particularly problematic in regions characterized by low soil fertility, limited crop rotation and intensive seasonal bean production.

Bean root health is an essential component in managing abiotic stresses as root pathogens aggravate problems of drought or phosphorus acquisition by restricting root systems. Improving the levels of root rot resistance is a key element in the successful development of drought tolerance in beans. For example, *Macrophomina* is a major problem under conditions of terminal drought (Frahm et al., 2004), whereas *Rhizoctonia and Fusarium* are major root pathogens in the regions where intermittent drought occurs (Navarrete-Maya et al., 2002). Cultivars such as Pinto Villa with resistance to intermittent drought that occurs in the Mexican highlands are also recognized for resistance to root rot (Acosta-Gallegos et al., 1995), suggesting that selection for drought tolerance under local

conditions may enhance root rot resistance. Likewise, BAT 477 with resistance to terminal drought is also recognized as a source of resistance to *Macrophomina*. Inheritance of resistance in BAT 477 is controlled by two complementary dominant genes (*Mp-1*, *Mp-2*) that segregated into discrete nine resistant: seven susceptible categories following greenhouse inoculations with a single isolate of *M. phaseolina* (Olaya et al., 1996). In addition to the *Mp-1*, *Mp-2* resistance genes, quantitative resistance conditioned by four QTL with relatively minor effect (13–19%) were reported in the Dorado/XAN 176 mapping population (Miklas et al., 1998b). Two of the larger-effect QTL that expressed across environments were located within resistance gene clusters (Figure 1) on linkage groups B4 and B7 (Miklas et al., 2000c). Although, Mayek-Pérez et al. (2001) reported a similar inheritance of resistance in BAT 477, lack of map integration and validation studies of the *Mp* genes- and QTL-linked markers in additional populations has restricted use of the markers in breeding for resistance to charcoal rot.

The widespread nature and importance of *F. solani* as the predominant root rot pathogen in common bean emphasizes the need for effective control through the development of resistant cultivars (Boomstra & Bliss, 1977; Schneider et al., 2001; Chowdbury et al., 2002; Navarro et al., 2003). However, complex inheritance combined with genetic incompatibility between gene pools have limited attempts to incorporate Fusarium root rot resistance into large-seeded Andean bean cultivars, despite the existence of extensive information on sources of resistance in the Middle American gene pool (Beebe & Bliss, 1981; Wallace & Wilkinson, 1975).

Indirect selection for Fusarium root rot resistance based on markers linked to the resistance QTL would facilitate improvement of root rot resistance, as direct field selection is laborious and destructive sampling is needed to identify resistance. Over 30 QTL, many minor in effect, associated with root rot resistance have been reported in RIL populations derived from four resistance sources. Note that only the QTL with larger effects were included in Figure 1. Sixteen QTL for *Fusarium* root rot resistance were identified in a RIL population derived from susceptible cultivar, Montcalm crossed with resistant line FR266 (Schneider et al., 2001); two QTL were identified in a RIL population derived from susceptible cultivar AC Compass crossed to resistant line NY2114-12, (Chowdbury et al., 2002); six QTL were identified in a RIL population derived from susceptible snap bean cultivar Eagle crossed with resistant line Puebla 152 (Navarro et al., 2003); and nine QTL were identified in two inbred backcross line populations derived from the susceptible cultivars Red Hawk and C97407 crossed to resistant line Negro San Luis (Román-Avilés & Kelly, 2005). A single large-effect QTL was detected by Román-Avilés and Kelly (2005) on B5 that accounted for up to 53% of the phenotypic variation for *Fusarium* root rot resistance that could be backcrossed into susceptible germplasm using MAS. A second QTL on B5 that explained up to 30% of the variation for resistance was linked to one of the markers, previously identified as associated to root rot resistance (Schneider et al., 2001). Most of the QTL located on linkage groups B2 and B3 of the integrated bean map (Freyre et al., 1998) were close to a region where defense response genes *Pgip*, and *ChS* and pathogenesis-related proteins, *PvPR-1* and *PvPR-2*, have been identified (Schneider et al., 2001). The detection of QTL in the same genomic regions as previously reported QTL for root rot resistance would suggest that different resistance sources might possess similar genes or resistance mechanisms associated with known defense response genes in *P. vulgaris*.

Other than the dominant monogenic resistance to Fusarium wilt (Cross et al., 2000) mapped to B10 (Fall et al., 2001), the inheritance of resistance to other root rot pathogens (Rhizoctonia, Pythium, Aphanomyces, etc.) have not been studied extensively nor have resistance genes or QTL been identified.

Rust

The highly variable nature of the rust pathogen, causal organism *Uromyces appendiculatus*, and the rapid breakdown of major gene resistance present in bean cultivars has challenged bean breeders working to develop durable resistance to bean rust. Pyramiding different resistance genes and mechanisms (specific, adult plant, slow rusting, reduced pustule size, and pubescence) should prolong the life of a bean cultivar by creating a more durable resistance complex (Mmbaga et al., 1996). The importance of such resistance gene pyramids was observed in Honduras. Bean lines carrying the broadly effective *Ur-11* resistance gene of Middle American origin were infected by a newly identified rust pathotype (Race 108) (Stavely et al., 1997), whereas lines possessing the hypostatic *Ur-4* resistance gene of Andean origin in addition to *Ur-11* were not infected (Mmbaga et al., 1996).

Nine major rust resistance genes *Ur-3*, *Ur-4*, *Ur-5*, *Ur-6* (Park et al., 2004), *Ur-7* (Park et al., 2003), *Ur-9* (Jung et al., 1998), *Ur-11*, *Ur-12* (Jung et al., 1998), and

Ur-13 (Mienie et al., 2005) and four unnamed genes, one each in breeding line BAC 6 (Jung et al., 1996) and Ouro Negro (Corrêa et al., 2000; Faleiro et al., 2000) and two in Dorado (Miklas et al., 2000c), have been characterized, tagged (Miklas, 2005) and mapped (reviewed by Kelly et al., 2003 and Miklas et al., 2002b). Note *Ur-12* conditions adult plant resistance whereas the other genes express specific resistance (Jung et al., 1998). Based on map location (Figure 1) and previous inheritance studies, the existence of gene clusters appears to be more common for rust than for anthracnose resistance genes in bean. For instance, Stavely (1984) showed that resistance to individual rust races in the bean line B-190 (*Ur-5* gene) is conditioned by single dominant genes linked in coupling that appear to be inherited as a complex linkage block. The apparent linkage of an additional unnamed gene from the cultivar Dorado suggests that the *Ur-5* genomic region on B4 may contain an even greater complex of linked genes than previously considered.

The tight linkage between *Ur-3* and *Ur-11* and apparent linkage of a different unnamed gene from Dorado is indicative of another rust resistance gene block on B11 (Figure 1). The five host differential cultivars Aurora, NEP 2, MEX 235, Ecuador 299, and 51051 are considered to possess *Ur-3* but with slightly different reaction profiles to a common set of rust races (Pastor-Corrales, personal communication, 2004). This observation is consistent with *Ur-3* being comprised of a block of resistance genes where differential genotypes carry different members of the linkage block. In addition, linkage group B11 possesses three other independent genes *Ur-6*, *Ur-7*, and an unnamed gene from BAC 6. Interestingly, Andean rust and anthracnose resistance genes co-localize (Kelly & Vallejo, 2004; Kelly et al., 2003; Miklas et al., 2002b) on linkage groups B1 (*Co-1* with *Ur-9*), whereas Middle American genes *Ur-5* with Co-3/*Co-9* and *Ur* gene from Ouro Negro with *Co-10* co-localize on B4, and *Ur-3* and others co-localize with *Co-2* on B11, suggesting that these genes are derived from common ancestral gene sequences. In our project (PNM), the Middle American genes *Ur-3*, *Ur-5* and *Ur-11* are being deployed by MAS into bean cultivars for East Africa because they are highly effective against the Andean races of the rust pathogen that predominate there (Liebenberg, personal communication, 2005). More effort to pyramid resistance in new cultivars is needed as too much emphasis is being placed on the use of the single *Ur-3* gene in North America despite the multigene pyramids available in breeding lines in three major US commercial bean seed classes (Pastor-Corrales, 2003).

White mold

White mold caused by *Sclerotinia sclerotiorum* (Lib.) deBary is a major disease concern for bean growers in cool sub-tropical and temperate climates where moist conditions prevail due to irrigation or rainfall. White mold is rated the most serious yield-limiting disease problem of beans in the United States. Fungicides are used to reduce the disease but are costly and timing of applications during the blossom period is critical for effective control. Combining genetic resistance with avoidance mechanisms, including upright and open plant structure, less dense canopies and branching patterns, elevated pod set, and reduced lodging (Schwartz et al., 1987), is the current breeding strategy for reducing white mold damage in dry bean (Kolkman & Kelly, 2002).

Genetic resistance is quantitatively inherited with low to moderate hertibility, as observed recently with the *P. vulgaris* resistance sources Bunsi, G 122, PC 50, and NY6020-4 (Ender & Kelly, 2005; Kolkman & Kelly, 2002; Miklas et al., 2001, 2003b, 2004; Park et al., 2001). Bunsi has been extensively used in breeding for white mold resistance in navy bean. The Bunsi source of resistance is conditioned by QTL on linkage groups B2, B3, B5, B7 and B8, with major-effect QTL residing on B2 and B7 (Kolkman & Kelly, 2003) and verified in multiple populations (Ender & Kelly, 2005). The QTL were found in regions of the genome associated with either plant architecture or general plant defense response genes, such as *PvPR-2* (Walter et al., 1990) and *Pgip* (Toubart et al., 1992) on B2, the *PvPR-1* gene on B3, and the seed lectins (Brambl & Gada, 1985) on B7. Quantitative trait loci for resistance on B7 were also significantly associated with yield, seed size, lodging and days to flower (Kolkman & Kelly, 2003). The resistance in Bunsi is ineffective in the straw test (Petzoldt & Dickson, 1996), which is a widely used greenhouse test used to evaluate beans for physiological resistance to white mold. Miklas et al. (2004) observed that stay-green trait where pods reach maturity but the plant remains green and physiologically active, was associated with resistance in a navy × pinto bean cross-segregating for Bunsi-derived resistance. A pinto bean line, AN-37 (released as USPT-WM-1; Miklas et al., 2005a), derived from this navy × pinto cross with stay-green stem trait possesses the major QTL from Bunsi that resides on B2.

Miklas et al. (2001) identified a major-effect QTL associated with white mold resistance from the genotype G122 on B7 that explained 38% of the variation in reaction for the straw test and 26% for the field. The QTL on B7 from Bunsi and G122 map at opposite ends of the linkage group, thus represent unique resistance sources. Two other sources of white mold resistance, PC-50 (Park et al., 2001) and NY6020-4 (Miklas et al., 2003b), have been analyzed in mapping populations. Three QTL from PC-50 mapped to linkage groups B4, B7, and B8. Two QTL from NY6020-4 were located on B6 and B8. The B7 and B8 QTL from PC-50 map to the same general location (Kelly et al., 2003) as the B7 QTL from G122 and the B8 QTL from NY6020-4, which suggests they may have resistance genes in common. Analysis of inbred backcross lines $BC_3F_{4:6}$ revealed that marker-assisted backcrossing was successful in transferring the B7 QTL from G122 and B8 QTL from NY6020-4 into susceptible pinto bean (Miklas & Bosak, 2004). The inbred backcross lines had similar yield, seed size, and seed appearance as the recurrent pinto parent but exhibited later maturity.

Recurrent selection solely for white mold resistance without regard for agronomic traits will undoubtedly result in lines with lower yield and later maturity, both undesirable traits. Kolkman and Kelly (2003) used a strategy, termed multi-trait bulking, to selectively map QTL conditioning resistance to white mold in a desirable high-yielding phenotype with commercially acceptable maturity. Conversely, Miklas et al. (2003b) used conventional bulked segregant analysis based solely on disease reaction phenotype that could result in the selective mapping of resistance QTL associated with undesirable traits such as late maturity.

The secondary gene pool (*P. coccineus*) exhibits potential for contributing resistance to common bean through interspecific hybridization. Monogenic (Schwartz et al., 2004) and polygenic models (Gilmore & Myers, 2004) of inheritance for the *P. coccineus* source of resistance have been observed. Dry bean lines with white mold resistance putatively derived from *P. coccineus* were released by Miklas et al. (1998a). The lines do not express the same level of resistance present in *P. coccineus* accessions, so further breeding directed toward introgressing the highest level of resistance possible from *P. coccineus* into common bean is needed. Given that resistance to white mold is a complexly-inherited trait, with low to moderate heritability and highly influenced by environmental factors that demands intensive field work to evaluate, MAS offers a promising approach to combine resistance QTL from diverse genomic regions (Figure 1) to improve overall resistance to white mold in common bean.

Biotic stresses: Insects

The genetics of insect resistance or tolerance in common bean is in general quantitative and polygenic, especially when compared to the genetics of specific disease resistance or tolerance. The mechanisms of resistance to insects in common bean can be divided as in other crops into antibiosis and antixenosis traits (Cardona & Kornegay, 1999). With a few exceptions, mainly having to do with biochemical traits such as seed protein, or morphological traits such as leaf hair density and trichome shape, the mechanisms underlying either type of resistance is unknown. Tolerance to insect attack is less well studied. The lack of information has made breeding for insect resistance or tolerance more complex than for disease resistance and as a result few cultivars have been bred specifically for insect resistance in common beans. However, host plant resistance is a promising component in an integrated cropping system for managing insect infestation in common bean and has the potential to reduce pesticide use and production costs while increasing on-farm yields.

Key to the utilization of insect resistance genes will be their further characterization and genetic tagging either as qualitative or quantitative traits. This review highlights the relatively few cases of gene or QTL tagging that have been completed for insect resistance and the potential that this endeavor has for success. The identification and mapping of insect resistance genes is expected to facilitate the development of molecular markers for MAS as has been achieved for disease resistance traits.

A large number of insects are pests of common beans but relatively few, including bean fly, bean pod weevil, bruchid weevils, leafhoppers, thrips and whiteflies, are of major importance and will be discussed later. Some of these pests are important worldwide, while others such as bean fly in Africa and bean pod weevil in Central America and Mexico are important regionally (Kornegay & Cardona, 1991). Other insect pests for which no resistance sources are known, such as chrysomelids, leafminers, pod borers or spider mites (Cardona & Kornegay, 1999) will not be discussed.

Apion

The bean pod weevil (*Apion godmani* Wagner) (Coleoptera: Curculionidae) is a destructive insect

pest of common beans grown in Mexico and Central America. Larvae of this pest burrow into immature seed inside developing pods causing yield loss and reduced seed quality (Cardona & Kornegay, 1999). Resistance is found in some race Jalisco landraces from Mexico such as Amarillo 153, Amarillo 154, Amarillo 155, Amarillo 169, Hidalgo 58, J-117, Puebla 36, Pinto Texcoco, Pinto 168 and Negro 150 (Garza et al., 1996, 2001).

Resistance to the bean pod weevil in common beans is thought to be controlled by two possible mechanisms: either antibiosis involving a hypersensitive response that encapsulates the oviposition sites, insect eggs or larvae within necrotic tissue; or antixenosis that affects the preference for oviposition sites (Garza et al., 2001). Epistasis between two independent dominant genes, *Agr* and *Agm*, has been hypothesized to control the hypersensitive response as shown from a partial diallel with seven of the resistant sources carried out at the INIFAP breeding station at Santa Lucia de Prias, Texcoco, Mexico (Garza et al., 1996). These are some of the few known major genes for insect resistance and the only known case of hypersensitive response to oviposition known in common bean, hypersensitivity being more typical of disease resistance than insect resistance in both this crop as well as other species (Fernandes, 1990; Yencho et al., 2000). The fact that a few genes control resistance may explain the observation that it was relatively straightforward to transfer resistance from Mexican landraces where it was found to new breeding lines with Central American seed types and high yields (Beebe et al., 1993).

Given the oligogenic nature of the resistance trait, CIAT in collaboration with INIFAP, have been developing SCAR molecular markers for Apion resistance using a recombinant inbred line population derived from the cross Jamapa × J-117, where J-117 is a resistance source for Apion and Jamapa is a susceptible black-seeded cultivar from Mexico. Preliminary results of a bulked segregant analysis are promising for the development of two or more SCAR markers linked to the resistance gene (Blair et al., 2003a). In addition to qualitative resistance based on the hypersensitive response which is stable across geographical areas and planting seasons, quantitative resistance affected by genotype × environment interaction has been observed for some sources of resistance from Guatemala (Garza et al., 1996). In the future, it would be interesting to analyze these quantitative resistance factors.

Bruchids

Bruchids (Coleoptera: Bruchidae), including the Mexican bean weevil [*Zabrotes subfasciatus* (Boheman)] and the bean weevil [*Acanthoscelides obtectus* (Say)], are the most common storage pests of common bean seed, causing an estimated 13% loss to bean crops worldwide (Kornegay & Cardona, 1991). *Zabrotes* is especially important in warm tropical regions below 1000 m altitude, while *Acanthoscelides* is more common in cooler climates. While *Zabrotes* is only found in storage, *Acanthoscelides* also lays its eggs on bean pods in the field. A special seed protein named arcelin, which was discovered in wild accessions of common beans from Mexico, provides high resistance to *Zabrotes* and slight resistance to *Acanthoscelides* (Schoonhoven et al., 1983; Osborn et al., 1988; Acosta-Gallegos et al., 1998). The arcelin gene has captured the imagination of many researchers as it represents a major gene resistance to an insect pest, which as mentioned earlier is uncommon; and is also one of the first and few examples of the utilization of the genetic resources found in wild common beans.

Arcelin and related proteins, including alpha amylase inhibitors and phytohemaglutinins (PHA) are all members of the APA family of seed proteins that provide resistance to bruchids through antibiosis by reducing the adult emergence, female fertility and insect growth and lifecycle (Osborn et al., 1988). These proteins are all synthesized only in the embryonic axis and cotyledons during seed formation. Arcelin is inherited as a monogenic trait and is located in a gene cluster on linkage group B4 (Figure 1) corresponding to the PHA/APA gene family (Osborn et al., 1986). Arcelin variants in wild common beans show evidence that the gene family arose from multiple duplication events which led to a complex locus. To date, seven variants of arcelin have been discovered and these variants are all highly similar but provide different levels of resistance (Sparvoli & Bollini, 1998). Within the allelic series the level of resistance is progressively lower in the variants ARC5 > ARC4 > ARC1 > ARC2 > ARC6 > ARC3 when in the background of the wild progenitor. However, in the cultivated background the alleles that provide the most resistance are ARC1 > ARC2 > ARC5 > ARC3 > ARC4 (Cardona & Kornegay, 1999). Differences in resistance level are thought to be due to sequence variability or carbohydrate content. Arcelin is known to be a partially dominant gene, which provides its highest level

of resistance to bruchids when in the homozygous form. Heterozygous Arc+/Arc− individual seeds are less resistant than Arc+/Arc+ seed. CIAT researchers have used the ARC1 variant widely in their breeding programs to create resistant breeding lines such as the RAZ breeding lines through backcrossing and gene transfer (Cardona et al., 1990). Despite this, no arcelin-derived bruchid-resistant cultivar has ever been released.

The method of selecting for arcelin-based resistance has been to assay for the protein in seed using a serological method which detects arcelin in small quantities of ground seed tissue (Kornegay et al., 1993). The process requires protein electrophoresis equipment and/or arcelin-specific antibodies. While technically somewhat demanding, arcelin-based selection of bruchid resistance has been very useful. For example, in the breeding of the RAZ lines at CIAT, two generations of backcrossing and selfing were generally sufficient to obtain resistant genotypes that were true to seed type when arcelin was evaluated during the backcrossing process (Cardona et al., 1990). The use of arcelin as a biochemical marker represents one of the first true uses of marker-based selection in common bean. However, limitations of the protein-based selection are that it is time-consuming and not compatible with DNA-based marker systems; therefore, new molecular markers are needed for the arcelin resistance gene.

Leafhoppers

Leafhoppers (*Empoasca* spp.; Homoptera: Cicadellidae) are common new-world pests of common bean that feed on phloem tissue, transmit viral diseases and cause stunting, downward leaf curling, yellowing and "hopper burn" (Kornegay & Cardona, 1991; Murray et al., 2004b). Hopper burn refers to necrosis or dessication of the leaf margins and growing tips that commonly follows a severe attack of the insect. The most common leafhoppers affecting common bean are: *E. fabae* (Harris) found in temperate regions of North America and *E. kraemeri* (Ross & Moore) found in tropical and sub-tropical regions of Caribbean, Central and South America. Resistance to *E. fabae* was once thought to be related to leaf hair pubescence especially to the presence of hooked trichomes which were postulated to capture adults or larval instars (Pillemer & Tingey, 1976); however, this has not been confirmed to be a significant antibiosis mechanism in the tropics where *E. kraemeri* is more frequent (Cardona & Kornegay, 1999). Apart from pubescence, indeterminate growth habit plants tend to have lower damage than determinate growth habit plants (Murray et al., 2001); however, this also is not thought to be a particularly effective tolerance mechanism and the heritability of tolerance is estimated to be low (Galwey & Evans, 1982; Kornegay & Temple, 1986). Another mechanism of resistance may be antixenosis, especially in the form of reduced feeding or probing by the insects (Kornegay et al., 1989), which is effective when combined with other forms of resistance or tolerance (Kornegay & Temple, 1986).

Quantitative trait loci studies have been undertaken for leafhopper resistance based on phenotyping of a recombinant inbred line population derived from a cross between a leafhopper-susceptible bean cultivar from Ontaria, Berna Dutch brown, and a leafhopper-resistant selection EMP 419 from CIAT (Murray et al., 2004a). Resistance to *Empoasca* in this population appeared to be purely quantitative with resistance to leaf burn controlled by a single QTL on linkage group B7 and resistance to leaf curl controlled by two QTLs, one adjacent to the leaf burn QTL on linkage group B7 and the other on linkage group B1 (Murray et al., 2004a; Figure 1). These observations confirm the need for recurrent selection and pyramiding of resistance sources to obtain high levels of *Empoasca* resistance (Cardona & Kornegay, 1999). Gamete selection with large population sizes has also been used to obtain *Empoasca* resistance in advanced lines of carioca beans (Singh et al., 1998). It will be interesting to determine if the inheritance of resistance is the same in Andean or determinate growth habit genotypes as it is in Mesoamerican or indeterminate growth habit genotypes used in the research of Murray et al. (2004a) and Singh et al. (1998).

Other insects (thrips, bean fly and whiteflies)

The melon thrips, *Thrips palmi* Karny (Thysanoptera: Thripidae), which spread recently to the Americas, can be an important pest on both dry and snap bean production especially in areas with problems of pesticide abuse and multiple alternate hosts for the insect pest (Cardona et al., 2002). Damage is caused by adults and larvae feeding on the leaves and growing tips of the plants and is visible on leaf ribs first, with foliage becoming silvery, leaves drying and drastic reduction in the number and size of pods under severe infection, affecting seed yield severely (Frei et al., 2003). As a result of population explosions of the insect, this pest

can decimate susceptible bean genotypes and result in total harvest loss.

Thrip resistance is not common among bean genotypes and in a large-scale screening the majority of genotypes have been shown to be highly susceptible (Cardona et al., 2002). Only a few mostly small-seeded Mesoamerican genotypes were found to have partial resistance or tolerance to the pest and resistance was not associated with maturity, growth habit, pubescence or seed color. In two further studies, Frei et al. (2003, 2004) found that resistance was due to a mix of tolerance as in the case of EMP 486, antixenosis as in the case of FEB 115 or combined resistance mechanisms including antibiosis for immature and adult survivorship and female longevity and total fecundity as in the case of Brunca (BAT 304), a released cultivar in Costa Rica and Cuba. In parallel, Frei et al. (2005) studied the inheritance of resistance in a recombinant inbred line population derived from the cross of BAT 881 × G 21212. Resistance was quantitative with some evidence of transgressive segregation with low to moderate heritabilities. In composite interval QTL analysis, the same authors identified a major QTL on linkage group B6 with additional QTLs on linkage groups B2, B3 and B8, many of which were located at regions of QTL for disease resistance genes (Figure 1). This study represented one of the first attempts to scrutinize QTLs for insect resistance in common bean based on a QTL mapping approach. The availability of a genome-wide microsatellite map for common bean (Blair et al., 2003b) will allow more such analysis to be conducted for insect resistance studies as was done by Frei et al. (2005).

Other important insect pests of common bean not mentioned earlier include the bean fly and whiteflies. The bean fly (*Ophiomyia phaseoli* Tryon and *O. spencerella* Greathead (Diptera: Agromyzidae) is one of the most serious pests of common beans in Africa and Asia. Some resistance has been found in the landrace varieties of common bean and scarlet runner bean (*P. coccineus*) (Kornegay & Cardona, 1991) but no genetic studies have been conducted on the inheritance of resistance or on genetic tagging of resistance genes. The whitefly species (Hemiptera: Aleyrodidae) found on beans include *Bemisia tabaci* (Gennadius) (biotype A and B), the sweetpotato or silverleaf whitefly, a vector of Geminiviridae plant viruses and *Trialeurodes vaporariorum* Westwood, the greenhouse whitefly, a major pest in horticultural areas. The distribution of each of these species depends on altitude and environment. Non-preference resistance to the sweetpotato whitefly is related to leaf hair pubescence with feeding and oviposition preference for genotypes with dense abaxial leaf hairs (Blair & Beaver, 1992). No genetic tagging studies have been conducted for this insect pest nor are markers developed for selection of resistance.

Pyramiding of insect resistance

Pyramiding of multiple insect resistance traits or of insect and disease resistance together has not been very frequent, but several attempts show that there is promise in this approach. Singh et al. (1998) were able to pyramid *Empoasca* resistance with resistance to four diseases in upright carioca-type beans through gamete selection, early generation testing and the use of EMP lines as sources; Bueno et al. (1999) incorporated multiple resistance to *Empoasca*, *Apion* and *Zabrotes* through single seed descent and evaluation of a large population of segregants. The identification and mapping of insect-resistance genes is expected to facilitate the development of resistant bean cultivars by using MAS but this will require the validation of some of the molecular markers discussed earlier and the wider application of others. MAS may be expected to expedite the pyramiding of major insect resistance genes to create multiple-resistant genotypes; however, since resistance to many insect pests is polygenic, pyramiding of resistance to several insect pests in a single genotype will remain a challenge.

Abiotic stresses

Abiotic stress resistance is by its nature more complex physiologically, is typically subject to large environmental effects and has been less well studied than biotic stress resistance in common bean (Rao, 2001). Therefore, compared to pest and disease resistance, much less is known about genetics of resistance to abiotic constraints or physiological stress. Plant response to one stress may be conditioned by the presence or absence of other stresses. For example, inadequate soil fertility or compacted soil structure may reduce root growth and thus limit the potential of the plant to express drought resistance. Abiotic stress resistance is typically governed by polygenic inheritance and may be conditioned by multiple, interacting mechanisms. These and other factors make abiotic stress resistance especially difficult to study, both physiologically and genetically.

Yet abiotic stress tolerance may be the key to improving yields of common bean in both stressed and unstressed environments if experience in other crops

is an indicator (Beebe et al., 2004). In both maize and soybean, improved yields in irrigated fields in Nebraska were associated with increased tolerance of higher plant densities (Specht et al., 1999), which is essentially resistance to lower per plant availability of resources (water, light and nutrients). Rao (2001) has summarized work on abiotic stress in common bean. At CIAT an integrated approach to abiotic stress tolerance that includes field and greenhouse physiological analysis, genomics and gene tagging is being pursued (Ishitani et al., 2004).

Two broad approaches can be described in the application of molecular markers to the development of stress-resistant bean cultivars. The first is focused on the use of yield as the criterion for stress resistance. Quantification of stress response to identify QTL for resistance is often based on the comparison of crop yield in stressed and unstressed treatments. The statistical tools to estimate resistance based on these two treatments vary widely, including: geometric means, percentage loss in yield and deviation from regression of stressed yields on unstressed yields (Singh, 1995; Beebe et al., 1997; Schneider et al., 1997a; Ramirez-Vallejo & Kelly, 1998). The method used will have direct implications for the estimated values of resistance and therefore, for the statistical analysis and the identification of QTL. The simplest way of overcoming this problem is to perform QTL analysis for stressed and unstressed yields independently to determine what QTL are specific for yield under abiotic stress, and therefore can be considered to be QTL for stress resistance.

The second approach to identifying markers for stress resistance is to seek genes or QTL for specific stress resistance traits or mechanisms. Alternatively, gene-based selection can utilize gene sequences of candidate genes as markers. These approaches are more focused and avoid some of the complexity of yield and its multiple causes and interactions. However, both approaches require prior, reliable physiological studies to demonstrate the positive contribution of either traits or genes to yield under stress across multiple environments. Molecular genetics (including QTL analysis) must interact with physiology to quantify the contributions of different genes, their respective mechanisms and the interactions of mechanisms. While the long-term objective of marker studies is developing systems for MAS, in the shorter term they can also shed light on plant physiology per se (Patterson, 1995).

Among the most important abiotic constraints limiting bean production are drought, phosphorus deficiency and nitrogen deficiency due to poor nitrogen fixation (Rao, 2001). An estimated 60% of bean production in Latin America and Africa suffers from phosphorus deficiency, although this is less of a constraint in temperate regions. Similarly, a majority of bean production areas in the tropics are affected by terminal or intermittent drought. In Africa alone, it is estimated that phosphorus deficiency, nitrogen deficiency and drought account for yield losses annually of 300–400 thousand MT each (Wortmann et al., 1998). It is within this context that we will review the inheritance of resistance to these three major abiotic stresses.

Drought

Local adaptation is an important component of drought resistance, as evidenced by a common set of genotypes evaluated in several countries in the 1980s (White, 1987). Conventional genetic studies of White et al. (1994) suggested that drought-resistant genotypes available at that time and selected respectively in the Mexican highlands and in Colombia did not adapt in the other environment, and that the value of drought resistance sources as parents was closely associated with the yield of the parent in the given environment. If the component of local adaptation is greater than that of drought tolerance per se, it suggests that even if genes or QTL are identified, these could be limited in expression if local adaptation dwarfs their effect. On the other hand, as breeding increases levels of resistance and "drought-specific" genes are accumulated in elite lines, the drought resistance component might become as important as local adaptation. This could lead to more stable expression of drought tolerance across sites and regions.

Rooting pattern, especially greater root length in lower soil strata, is an important drought resistance mechanism (Sponchiado et al., 1989). More recently, the capacity to partition a greater proportion of carbohydrate to seed under stress has emerged as an important trait (Rao, 2001). G 21212, a black-seeded accession from Colombia, has an unusual capacity to set pods and fill seeds under stress, and its genes have been unique enough to warrant genetic studies which are underway at CIAT (Beebe et al., 1999; Blair et al., 2002). Many other traits have been examined but none appear to play a prominent role in resistance to drought in common bean (Rao, 2001).

Phenotypic selection has been practiced with considerable success to improve drought tolerance. BAT 477, a race Mesoamerica breeding line, was identified in CIAT-Colombia as superior in drought resistance in

lowland tropical environments, but without prior directed breeding for this trait (White et al., 1994). Terán and Singh (2002) reported that in this same environment race Durango germplasm from the semiarid highlands of Mexico possessed the best drought resistance among landrace germplasm, but that even better lines were derived from a double cross combining race Durango (Guanajuato 31) and race Mesoamerica (BAT 477). The best line from this combination was SEA 5. Frahm et al. (2004) likewise reported that a superior black-seeded line for tropical environments, L88-63, was selected from a simple cross in which one of the parents was derived from a combination of Durango and Mesoamerica races. This line was superior in both drought-stressed and unstressed conditions and outyielded SEA 5. Further work at CIAT with interracial parental material and combining the deep rooting trait with improved seed filling also produced lines yielding as much as 50% more than SEA 5 (Ishitani et al., 2004). Thus, combining races Durango and Mesoamerica has been a consistent source of improved drought resistance for lowland tropical environments, and additional cycles of breeding have served to refine these combinations, probably resulting in more effective introgression of Durango genes to lowland race Mesoamerica. In highland environments of Mexico, Pinto Villa (Acosta-Gallegos et al., 1995) has proven superior. This cultivar combines race Durango with Andean germplasm.

Given the consistent success resulting from the introgression of genes from race Durango to race Mesoamerica or vice versa, this may be a good opportunity for the application of MAS, since race-specific polymorphism is reasonably common. The potential to select drought tolerance with QTL analysis and MAS was investigated by Schneider et al. (1997b) in seven environments in Michigan and highland Mexico. Using RAPD, four markers for QTL were identified in one population and five in a second population. Selection based on MAS was effective under severe drought in Michigan but not for moderate drought in Mexico. Genotype × environment interaction apparently affected the expression of QTL and furthermore, genome coverage was incomplete and some unidentified QTL might have determined yield in the Mexican environments. Additional preliminary drought QTL have been identified for the BAT 477 source under non-irrigated conditions at CIAT (Blair et al., 2002). Dehydration-responsive element binding protein (DREB) genes have been identified in beans but their significance to drought tolerance remains to be demonstrated (Galindo et al., 2003). If these prove to have drought-resistance functions in common bean, then gene-based MAS might be feasible (Ishitani et al., 2004).

Low phosphorus

Genetic variability in common bean has been widely documented for the capacity to produce grain in conditions of low soil phosphorus availability (Rao, 2001; Beebe et al., 1997). Lynch and Beebe (1995) described a theoretical basis for these differences based on differences in root structure, and some implications of differences in root structure have been confirmed or discarded through the study of genetic mapping populations and QTL analysis. For example, Liao et al. (2004) showed that under phosphorus stress the basal roots of bean accession G 19833 could reorient to explore more shallow soil strata where phosphorus is concentrated. It was possible to map QTL from G 19833 that were responsible for this reaction on linkage groups B1, B2, B3, B4, B5, B6, B10 and B11 in a recombinant inbred line population derived from the cross DOR 364 × G 19833. While several QTL for phosphorus uptake from G 19833 were on different linkage groups, two were linked to QTL for the shallow rooting trait on linkage groups B4 and B11. Other important QTL for phosphorus uptake derived from DOR 364 were found on B9 and B10 and were not associated with basal roots growth angle or length (Yan et al., 2004). Thus, phosphorus absorption in common bean is a complex trait with multiple mechanisms (Yan et al., 1995a,b, 1996).

Root acid exudation is another trait that may underlay phosphorus uptake efficiency with QTL for this trait identified in the DOR 364 × G 19833 mapping population on linkage groups B4, B5 and B10 in the same locations as some of the QTL mentioned earlier for phosphorus uptake efficiency (Yan et al., 2004). In this same study, total root acid exudation was correlated with basal root hair density and length but a greater number of QTL were found for basal root hair density and length on linkage groups B1, B3, B9, B10 and B11 (Yan et al., 2004). In contrast, higher levels of leaf phosphatase might be expected to improve phosphorus use efficiency by facilitating better remobilization of phosphorus within the plant, but a major gene difference in this trait did not alter phosphorus use efficiency in common bean (Yan et al., 2001).

Preliminary evaluation in another cross combination has shown that the accession G 21212 has important QTL for yield under low P conditions (Beebe et al., 1999; Blair et al., 2002). Its mechanism appears to be

associated in large degree not with acquisition but use of phosphorus, based on greater grain production per unit of phosphorus in the plant, and the QTL from this genotype can be considered to be the best candidates at present to use in selection for and development of MAS for low phosphorus adaptation, given the magnitude of their effects. Whether MAS or phenotypic selection will be more efficient for this trait and the others described earlier depends on how routine phenotypic screens or marker assays might become relative to each other. Another consideration is that root architectural traits may not be so unique to a given parent as to warrant developing markers specifically for that parent. Roots are highly plastic and there may be multiple ways to produce a desirable phenotype. If this proves to be the case, then a phenotypic screen may be as effective as MAS. However, from the results reviewed in this paper it can be concluded that QTL analysis has been very useful in determining which root traits contribute to phosphorus uptake.

Symbiotic nitrogen fixation (SNF)

Genetics and breeding for SNF has been investigated extensively and was reviewed by Bliss (1993) and by Snoeck et al. (2003). The potential to improve SNF has been amply demonstrated in common bean (Bliss, 1993; Barron et al., 1999). However, SNF improvement typically does not form a part of routine cultivar improvement programs, and incorporating selection criteria for SNF such as nodule mass, nitrogenase activity and xylem ureide content into breeding schemes while attending to other breeding objectives remains a challenge. If MAS for SNF could be integrated into breeding programs that are already practicing MAS for other traits, this would avoid the necessity of additional phenotypic selection methodologies purely for nitrogen or nodule determination.

As many as six chromosomal regions for degree of nodulation were identified on an RFLP map based on a cross of BAT 93 and Jalo EEP558, which are Mesoamerican and Andean genotypes respectively (Tsai et al., 1998). However, no effort was made in this study to associate QTL with either N fixation or yield, and thus their utility to genetic improvement requires confirmation. ESTs from nodules have been developed (VandenBosch & Stacey, 2003) and gene-based MAS would appear to have potential in the long run, as SNF genes are being identified in many species including model legumes *Lotus japonicus* (Webb et al., 1999) and *Medicago trunculata* (Gyorgyey et al., 2000). The challenge in this case appears to lie in identifying the key genes that explain differences in the field among a great array of expressed genes.

In tropical soils, low availability of soil phosphorus often limits SNF, and genetic variability for the ability to fix N at low P has been reported (Vadez et al., 1999). In subsequent trials, BAT 477 displayed this trait (CIAT, unpublished) and also exhibits superior nitrogen fixation under optimal conditions (Kipe-Nolt et al., 1993) and under drought conditions (Castellanos et al., 1996). In this regard, BAT 477 is an unusual genotype and its genes seem to merit tagging and reselection. BAT 477 may possess SNF genes that are active over multiple stress conditions because they are less sensitive to stimuli that result in down-regulation (CIAT, 1998). Gene tagging has been pursued based on nitrogen accumulation in a controlled greenhouse environment (CIAT, unpublished). Some QTL that were expressed at low phosphorus also contributed to SNF at high phosphorus. A microsatellite marker associated with QTL for nitrogen accumulation is being introgressed from BAT 477 into race Durango cultivars in use in the highlands of Mexico, with expectations of improving fixation under conditions of water deficit. If this effort is successful, it may open new opportunities for SNF improvement. If MAS can become the primary selection criterion of SNF and can be coordinated with selection for other traits, MAS could stimulate more directed selection for SNF and thus permit capitalizing on the vast body of knowledge that exists about genetic variability in SNF in common bean.

Conclusions

For sustained development of improved bean cultivars, researchers need to continue to (i) gain knowledge about the biotic and abiotic stresses of economic importance in production regions around the world; (ii) identify, share, and preserve sources of resistance to the important stresses; (iii) develop faster and more reliable screening procedures for both direct selection (phenotypic) and MAS of resistance traits; (iv) gain a better understanding of the inheritance and mechanisms of resistance especially for complex stresses; (v) conduct molecular genetics and genomic studies relevant to gaining a better understanding of the genetics and physiology of resistance; (vi) translate molecular and genomics information obtained into tools useful for marker-aided breeding; and (vii) integrate marker-aided breeding to compliment classical breeding on a

case by case basis. Given the recent deployment of MAS in plant breeding, careful consideration of the benefits and limitations of implementing MAS is warranted, such as closely monitoring its efficiency and utility in specific applications.

One disadvantage of MAS is that it usually commits a breeder to using a specific parental source of a trait in which the marker for a gene is expressed and which is polymorphic in relation to other parental materials. This may be restrictive and exclusive of other equally useful sources of the trait. Similarly, a desired trait might result from multiple genetic combinations. For example, roots are genetically complex so several different genetic combinations may be needed to produce a desirable root phenotype. Phenotypic selection on the other hand permits selecting a desired phenotype from any promising parental source, or that can result from different combinations of genes. Thus, a breeder will want to consider whether it is more advantageous to practice MAS using a specific source of a trait, or to use phenotypic selection that admits using alternative sources of the trait. MAS is especially useful when the tagged gene is truly unique and there are few alternatives to obtain the desired phenotype, as in the case of monogenic disease resistance.

Phenotypic selection has led to significant advances in abiotic stress resistance, especially in drought resistance. Breeders must decide when and how MAS can contribute to more efficient selection schemes. Can MAS contribute additional benefits over phenotypic selection? This will depend on the following:

(i) Identification of QTL or genes with a significant effect and that are sufficiently unique that developing markers based on a given parent outweighs any disadvantage of limiting one's crosses to the use of that parent. However, the gene pool and race structure of common bean (Singh et al., 1991; Beebe et al., 2000) lend themselves to polymorphism and in cases where introgression among pools or races is practiced, then MAS may be very useful.

(ii) Confirmation of the value of QTL over environments. While the investment in confirmation trials cannot exceed what would be spent on phenotypic selection, some confidence in the wide value of QTL is necessary.

(iii) Efficient selection systems. To date, all selection systems that have been employed in common bean are gel based. We know of no case of using RFLP in MAS, and most applications employ SCAR developed from RAPD, although a few SSR are in application. In all these cases, rustic markers that can function with poor-quality DNA from "quick-and-dirty" extraction techniques are a must for scaling up selection. Once MAS systems are set up for other traits, the cost per trait will drop, making MAS more attractive. More markers also increase chances to deploy multiplexing, either in PCR amplification or on gels to reduce costs (Figure 2). Opportunities clearly exist in common bean to test these and other theories now that a wide array of markers linked to many traits of economic importance are available to bean breeders.

References

Abawi, G.S. & M.A. Pastor-Corrales, 1990. Root rots of beans in Latin America and Africa: Diagnosis, research methodologies, and management strategies. Centro Internacional de Agricultura Tropical (CIAT), Cali, Colombia, 114 p.

Acevedo-Román, M., A. Molina-Castañeda, J.C. Angel Sánchez, C.G. Muñoz & J.S. Beaver, 2004. Inheritance of normal pod development in bean golden yellow mosaic resistant common bean. J Am Soc Hort Sci 129: 549–552.

Acosta-Gallegos, J.A., R. Ochoa-Marquez, M.P. Arrieta-Montiel, F. Ibarra-Perez, A. Pajarito-Ravelero & I. Sanchez-Valdez, 1995. Registration of 'Pinto Villa' common bean. Crop Sci 35: 1211.

Acosta-Gallegos, J.A., C. Quintero, J. Vargas, O. Toro, J. Tohme & C. Cardona, 1998. A new variant of arcelin in wild common bean *Phaseolus vulgaris* L. from southern México. Genet Resources Crop Evol 45: 235–242.

Adam-Blondon, A.F., M. Sevignac, H. Bannerot & M. Dron, 1994. SCAR, RAPD, and RFLP markers linked to the dominant gene (Are) conferring resistance to anthracnose. Theor Appl Genet 88: 865–870.

Aggarwal, V.D., M.A. Pastor-Corrales, R.M. Chirwa & R.A. Buruchara, 2004. Andean beans (*Phaseolus vulgaris* L.) with resistance to the angular leaf spot pathogen (*Phaeoisariopsis griseola*) in southern and eastern Africa. Euphytica 136: 201–210.

Ariyarathne, H.M., D.P. Coyne, G. Jung, P.W. Skroch, A.K. Vidaver, J.R. Steadman, P.N. Miklas & M.J. Bassett, 1999. Molecular mapping of disease resistance genes for halo blight, common bacterial blight, and bean common mosaic virus in a segregating population of common bean. J Am Soc Hort Sci 124: 654–662.

Balardin, R.S. & J.D. Kelly, 1998. Interaction among races of *Colletotrichum lindemuthianum* and diversity in *Phaseolus vulgaris*. J Am Soc Hort Sci 123: 1038–1047.

Barron, J.E., R.S. Pasini, D.W. Davis, D.D. Stuthman & P.H. Graham, 1999. Response to selection for seed yield and nitrogen (N2) fixation in common bean (*Phaseolus vulgaris* L.). Field Crops Research 62: 119–128.

Bassett, M.J., 2004. List of genes – *Phaseolus vulgaris* L. Annu Rep Bean Improv Coop 47: 1–24.

Beaver, J.S., J.M. Osorno, F.H. Ferwerda & C.G. Muñoz Perea, 2005. Registration of bean golden yellow mosaic virus resistant germplasms PR9771-3-1, PR0247-49 and PR0157-4-1. Crop Sci 45: (in press).

Beaver, J., J.C. Rosas, J. Myers, J. Acosta, J.D. Kelly, S. Nchimbi-Msolla, R. Misangu, J. Bokosi, S. Temple, E. Arnaud-Santana & D.P. Coyne, 2003. Contributions of the Bean/Cowpea CRSP to cultivar and germplasm development in common bean. Field Crops Res 82: 87–102.

Beebe, S., 1994. Breeding for resistance to bean golden mosaic virus: History and perspective. In F. Morales (Ed.), Bean Golden Mosaic: Research Advances, p. 148–150. CIAT, Cali, Colombia.

Beebe, S.E. & F.A. Bliss, 1981. Root rot resistance in common bean germplasm of Latin American origin. Plant Dis 65: 485–489.

Beebe, S., C. Cardona, O. Díaz, F. Rodríguez, E. Mancia & S. Ajquejay, 1993. Development of common bean (*Phaseolus vulgaris* L.) lines resistant to the bean pod weevil, *Apion godmani* Wagner, in Central America. Euphytica 96: 83–88.

Beebe, S., J. Lynch, N. Galwey, J. Tohme & I. Ochoa, 1997. A geographical approach to identify phosphorus-efficient genotypes among landraces and wild ancestors of common bean. Euphytica 95: 325–336.

Beebe, S.E. & M. Pastor-Corrales, 1991. Breeding for disease resistance. In A. van Schoonhoven & O. Voysest (Eds.), Common Beans: Research for Crop Improvement, p. 561–617. CAB International, Wallingford, UK/CIAT, Cali, Colombia.

Beebe, S., I. Rao, H. Teran & C. Cajiao, 2004. Breeding concepts and approaches in food legumes: The example of the common bean. Abstract of paper presented at the "Second National Workshop on Food and Forage Legumes" Addis Ababa, Ethiopia, 22–26 September 2003.

Beebe, S., A. Velasco & F. Pedraza, 1999. Marcaje de genes para rendimiento en condiciones de alto y bajo fósforo en las accesiones de frijol G21212 y BAT 881. Poster presented in the VI Reunião Nacional de Pesquisa de Feijão, Salvador, Brazil, 21–26 November 1999.

Beebe, S., P.W. Skroch, J. Tohme, M.C. Duque, F. Pedraza & J. Nienhuis, 2000. Structure of genetic diversity among common bean landraces of Mesoamerican origin based on Correspondence Analysis of RAPD. Crop Sci 40: 264–273.

Bennett, C.W., 1971. The curly top disease of sugarbeet and other plants. Am Phytopathol Soc Monogr No. 7.

Bianchini, A., V. Moda-Cirino, N.S. Fonseca & F.F. Toledo, 1994. Genética de resistência ao vírus do mosaico dourado do feijoeiro. (Abstr) Fitopatol Bras 19: 329.

Blair, M.W. & J.S. Beaver, 1992. Resistance to the sweetpotato whitefly (*Bemisia tabaci*), the vector of bean golden mosaic virus in dry beans. Annu Rpt Bean Improv Coop 35: 154–155.

Blair, M.W., C. Cardona, C. Muñoz, H.F. Buendía & R. Garza, 2003a. Development of SCAR markers for Apion resistance. CIAT Annual Report, pp. 158–159. Cali, Colombia.

Blair, M.W., M.C. Muñoz & S.E. Beebe, 2002. QTL analysis of drought and abiotic stress tolerance in common bean RIL populations. In: Annual Report, Biotechnology Research Project, pp. 68–72. CIAT, Cali, Colombia.

Blair, M.W., F. Pedraza, H.F. Buendia, E. Gaitán-Solís, S.E. Beebe, P. Gepts & J. Tohme, 2003b. Development of a genome-wide anchored microsatellite map for common bean (*Phaseolus vulgaris* L.) Theor Appl Genet 107: 1362–1374.

Bliss, F.A., 1993. Breeding common bean for improved nitrogen fixation. Plant Soil 152: 71–79.

Boomstra, A.G. & F.A. Bliss, 1977. Inheritance of resistance to *Fusarium solani* f.sp. *phaseoli* in beans (*Phaseolus vulgaris* L.) and breeding strategy to transfer resistance. J Am Soc Hort Sci 102: 186–188.

Brambl, R. & W. Gada, 1985. Plant seed lectins disrupt growth of germinating fungal spores. Physiol Plant 64: 402–408.

Broughton, W.J., G. Hernandez, M. Blair, S. Beebe, P. Gepts & J. Vanderleyden, 2003. Bean (*Phaseolus* spp.)-model food legumes. Plant Soil 252: 55–128.

Bueno, J.M., C. Cardona & C.M. Quintero, 1999. Comparison between two improvement methods to develop multiple insect resistance in common bean (*Phaseolus vulgaris* L.). Revista Colombiana de Entomología 25: 73–78.

Busogoro, J.P., M.H. Jijakli & P. Lepoivre, 1999a. Identification of novel sources of resistance to angular leaf spot disease of common bean within the secondary gene pool. Plant Breed 118: 417–423.

Busogoro, J.P., M.H. Jijakli & P. Lepoivre, 1999b. Virulence variation and RAPD polymorphism in African isolates of *Phaeoisariopsis griseola* (Sacc.) Ferr., the causal agent of angular leaf spot of common bean. Eur J Plant Pathol 105: 559–569.

Caixeta, E.T., A. Borém, S. Azevedo Fagundes, S. Niestche, E.G. Barros & M.A. Moreira, 2003. Inheritance of angular leaf spot resistance in common bean line BAT 332 and identification of RAPD marker linked to the resistance gene. Euphytica 134: 297–303.

Campa, A., C. Rodríguez-Suárez, A. Pañeda, R. Giradles, & J.J. Ferreira, 2005. The bean anthracnose resístance gene Co-5 is located in linkage group B7. Annu Rep Bean Improv Coop 48: 68–69.

Cardona, C. & J. Kornegay, 1999. Bean germplasm resources for insect resistance. In: S.L. Clement, & S.S. Quisenberry (Eds.), Global Plant Genetic Resources for Insect-Resistant Crops. CRC Press, Boca Raton, FL.

Cardona, C., A. Frei, J.M. Bueno, J. Díaz, H. Gu & S. Dorn, 2002. Resistance to *Thrips palmi* (Thysanoptera: Thripidae) in beans. J Econ Entomol 95: 1066–1073.

Cardona, C., J.L. Kornegay, C.E. Posso, F. Morales & H. Ramírez, 1990. Comparative value of four arcelin variant in the development of dry bean lines resistant to the Mexican bean weevil. Entomol Exp Appl 56: 197–206.

Carvalho, G.A., T.J. Paula Jr., A.L. Alzate-Marin, S. Nietsche, E.G. Barros & M.A. Moreira, 1998. Herança da resistência da linhagem AND-277 de feijoeiro-comum à raça 63-23 de *Phaeoisariopsis griseola* e identificação de marcador RAPD ligado ao gene de resistência. Fitopatol Bras 23: 482–485.

Castellanos, J.Z., J.J. Peña-Cabrales & J.A. Acosta-Gallegos, 1996. 15N-determined dinitrogen fixation capacity of common bean (*Phaseolus vulgaris*) cultivars under water stress. J Agric Sci 126: 327–333.

Chowdbury, M.A., K. Yu & S.J. Park, 2002. Molecular mapping of root rot resistance in common bean. Annu Rep Bean Improv Coop 45: 96–97.

CIAT, 1998. Annual report of the bean project.

Corrêa, R.X., M.R. Costa, P.I. Good-God, V.A. Ragagnin, F.G. Faleiro, M.A. Moreira & E.G. Barros, 2000. Sequence characterized amplified regions linked to rust resistance genes in the common bean. Crop Sci 40: 804–807.

Corrêa, R.X., P.I. Good-God, M.L.P. Oliveira, S. Niestche, M.A. Moreira & E.G. Barros, 2001. Herança da resistência à mancha-angular do feijoeiro e identificação de marcadores moleculares flanqueando o loco de resistência. Fitopatol Bras 26: 27–32.

Coyne, D.P., J.R. Steadman, G. Godoy-Lutz, R. Gilbertson, E. Arnaud-Santana, J.S. Beaver & J.R. Myers, 2003. Contributions of the Bean/Cowpea CRSP to management of bean diseases. Field Crops Res 82: 155–162.

Cross, H., M.A. Brick, H.F. Schwartz, L.W. Panella & P.F. Byrne, 2000. Inheritance of resistance to Fusarium wilt in two common bean races. Crop Sci 40: 954–958.

Drijfhout, E., 1978. Genetic interaction between *Phaseolus vulgaris* and bean common mosaic virus with implications for strain identification and breeding resistance. Agricultural Research Reports, Vol. 872, pp. 1–98. Centre for Agriculture Publishing and Documentation, Wageningen, The Netherlands.

Elmer, J.S., G. Sunter, W.E. Gardiner, L. Brand, C.K. Browning, D.M. Bisaro & S.G. Rogers, 1988. Agrobacterium-mediated inoculation of plants with tomato golden mosaic virus DNAs. Plant Mol Biol Int J Mol Biol Biochem Genet Eng 10: 225–234.

Ernest, E.G. & J.D. Kelly, 2004. The Mesoamerican anthracnose resistance gene *Co-4*^{2} does not confer resistance in certain Andean backgrounds. Annu Rep Bean Improv Coop 47: 245–246.

Ender, M. & J.D. Kelly, 2005. Identification of QTL associated with white mold resistance in common bean. Crop Sci 45: (in press).

Faleiro, F.G., W.S. Vinhadelli, V.A. Ragagnin, R.X. Corrêa, M.A. Moreira & E.G. Barros, 2000. RAPD markers linked to a block of genes confering rust resistance to the common bean. Genet Mol Biol 23: 399–402.

Fall, A.L., P.F. Byrne, J. Jung, D.P. Coyne, M.A. Brick & H.F. Schwartz, 2001. Detection and mapping of a major locus for fusarium wilt resistance in common bean. Crop Sci 41: 1494–1498.

Fernandes, G.W., 1990. Hypersensitivity: A neglected plant resistance mechanism against insect herbivores. Environ Entomol 19: 1173–1182.

Ferreira, C.F., A. Borém, G.A. Carvalho, S. Nietsche, T.J. Paula Jr., E.G. Barros & M.A. Moreira, 2000. Inheritance of angular leaf spot resistance in common bean and identification of a RAPD marker linked to a resistance gene. Crop Sci 40: 1130–1133.

Ferrier-Cana, E., V. Geffroy, C. Macadre, F. Creusot, P. Imbert-Bollore, M. Sevignac & T. Langin, 2003. Characterization of expressed NBS-LRR resistance gene candidates from common bean. Theor Appl Genet 106: 251–261.

Fourie, D. & L. Herselman, 2002. Breeding for common blight resistance in dry beans in South Africa. Annu Rep Bean Improv Coop 45: 50–51.

Fourie, D., P.N. Miklas & H.M. Ariyarathne, 2004. Genes conditioning halo blight resistance to races 1, 7, and 9 occur in a tight cluster. Annu Rep Bean Improv Coop 47: 103–104.

Frahm, M.A., J.C. Rosas, N. Mayek-Pérez, E. López-Salinas, J.A. Acosta-Gallegos & J.D. Kelly, 2004. Breeding beans for resistance to terminal drought in the lowland tropics. Euphytica 136: 223–232.

Frei, A., M.W. Blair, C. Cardona, S.E. Beebe, H. Gu & S. Dorn, 2005. QTL mapping of resistance to *Thrips palmi* Karny in common bean. Crop Sci 45: 379–387.

Frei, A., J.M. Bueno, J. Diaz-Montano, H. Gu, C. Cardona & S. Dorn, 2004. Tolerance as a mechanism of resistance to *Thrips palmi* in common beans. Entomol Exp Appl 112: 73–80.

Frei, A., H. Gu, J.M. Bueno, C. Cardona & S. Dorn, 2003. Antixenosis and antibiosis in the resistance of bean plants to *Thrips palmi* Karny (Thysanoptera: Thripidae). J Econ Entomol 96: 1577–1584.

Freyre, R., P. Skroch, V. Geffroy, A.F. Adam-Blondon, A. Shirmohamadali, W. Johnson, V. Llaca, R. Nodari, P. Pereira, S.M. Tsai, J. Tohme, M. Dron, J. Nienhuis, C. Vallejos & P. Gepts, 1998. Towards an integrated linkage map of common bean. 4. Development of a core map and alignment of RFLP maps. Theor Appl Genet 97: 847–856.

Galindo, L., M.W. Blair, S. Beebe, M. Ishitani & J. Tohme, 2003. Development of a molecular marker: DREB for drought tolerance in common bean. In: Annual Report, Biotechnology Research Project, pp. 177–179. CIAT, Cali, Colombia.

Gálvez, G.E. & F.J. Morales, 1989. Whitefly-transmitted viruses. In: H.F. Schwartz & M.A. Pastor-Corrales (Eds.), Bean Production Problems in the Tropics, p. 379–408. CIAT, Cali, Colombia.

Galwey, N.W. & A.M. Evans, 1982. The inheritance of resistance to *Empoasca kraemeri* Ross and Moore in the common bean, *Phaseolus vulgaris* L. Euphytica 31: 933–952.

Garza, R., C. Cardona & S.P. Singh, 1996. In: heritance of resistance to the bean-pod weevil (*Apion godmani* Wagner) in common beans from Mexico. Theor Appl Genet 92: 357–362.

Garza, R., J. Vera, C. Cardona, N. Barbcenas & S.P. Singh, 2001. Hypersensitive response of beans to *Apion godmani* (Coleoptera: Curculionidae). J Econ Entomol 94: 958–962.

Geffroy, V., F. Creusot, J. Falquet, M. Sévignac, A.F. Adam-Blondon, H. Bannerot, P. Gepts & M. Dron, 1998. A family of LRR sequences at the *Co-2* locus for anthracnose resistance in *Phaseolus vulgaris* and its potential use in marker-assisted selection. Theor Appl Genet 96: 494–502.

Geffroy, V., M. Sévignac, J. De Oliveira, G. Fouilloux, P. Skroch, P. Thoquet, P. Gepts, T. Langin & M. Dron, 2000. In: heritance of partial resistance against *Colletotrichum lindemuthianum* in *Phaseolus vulgaris* and co-localization of QTL with genes involved in specific resistance. Mol Plant Micr Inter 13: 287–296.

Geffroy, V., D. Sicard, J.C. De Oliveira, M. Sévignac, M. Cohen, P. Gepts, C. Neema, T. Langin & M. Dron, 1999. Identification of an ancestral resistance gene cluster involved in the coevolution process between *Phaseolus vulgaris* and its fungal pathogen *Colletotrichum lindemuthianum*. Mol Plant Micr Inter 12: 774–784.

Gepts, P., 1999. Development of an integrated linkage map. In: S.P. Singh (Ed.), Developments in Plant Breeding, Common Bean Improvement in the Twenty-First Century, pp. 53–91. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Gilmore, B. & J.R. Myers, 2004. A preliminary molecular map for *Phaseolus coccineus*. Annu Rep Bean Improv Coop 47: 87–88.

Guzmán, P., R.L. Gilbertson, R. Nodari, W.C. Johnson, S.R. Temple, D. Mandala, A.B.C. Mkandawire & P. Gepts, 1995. Characterization of variability in the fungus *Phaeoisariopsis griseola* suggests coevolution with the common bean (*Phaseolus vulgaris*). Phytopathology 85: 600–607.

Gyorgyey, J., J.D. Vaubert, J.I. Jimenez-Zurdo, C. Charon, L. Troussard, A. Kondorosi & E. Kondorosi, 2000. Analysis of *Medicago truncatula* nodule expressed sequence tags. Mol Plant Micro Inter 13: 62–71.

Haley, S.D., L. Afanador & J.D. Kelly, 1994a. Selection for monogenic pest traits with coupling and repulsion phase RAPD markers. Crop Sci 34: 1061–1066.

Haley, S.D., L. Afanador & J.D. Kelly, 1994b. Identification and application of a random amplified polymorphic DNA marker for the *I* gene (potyvirus resistance) in common bean. Phytopathology 84: 157–160.

Haley, S.D., L.K. Afanador, P.N. Miklas, J.R. Stavely & J.D. Kelly, 1994c. Heterogeneous inbred populations are useful as sources of near-isogenic lines for RAPD marker localization. Theor Appl Genet 88: 337–342.

Haley, S.D., P.N. Miklas, J.R. Stavely, J. Byrum & J.D. Kelly, 1993. Identification of RAPD markers linked to a major rust resistance gene block in common bean. Theor Appl Genet 86: 505–512.

Hangen, L.A. & M.R. Bennink, 2003. Consumption of black beans and navy beans (*Phaseolus vulgaris*) reduced azoxymethane-induced colon cancer in rats. Nutr Cancer 44: 60–65.

Hu, J. & B.A. Vick, 2003. Target region amplified polymorphism: A novel marker technique for plant genotyping. Plant Mol Biol Rep 21: 1–6.

Ishitani, M., I. Rao, P. Wenzl, S. Beebe & J. Tohme, 2004. Integration of genomics approach with traditional breeding towards improving abiotic stress adaptation: Drought and aluminum toxicity as case studies. Field Crop Res 90: 35–45.

Islam, F.M.A., K.E. Basford, R.J. Redden, C. Jara & S. Beebe, 2002. Patterns of resistance to angular leaf spot, anthracnose and common bacterial blight in common bean germplasm. Aust J Expt Agric 42: 481–490.

Johnson, W.C., P. Guzman, D. Mandala, A.B.C. Mkandawire, S. Temple, R.L. Gilbertson & P. Gepts, 1997. Molecular tagging of the *bc-3* gene for introgression into Andean common bean. Crop Sci 37: 248–254.

Johnson, E., P.N. Miklas, J.R. Stavely & J.C. Martinez-Cruzado, 1995. Coupling- and repulsion-phase RAPDs for marker-assisted selection of PI 181996 rust resistance in common bean. Theor Appl Genet 90: 659–664.

Jung, G., H.M. Ariyarathne, D.P. Coyne & J. Nienhuis, 2003. Mapping QTL for bacterial brown spot resistance under natural infection in field and seedling stem inoculation in growth chamber in common bean. Crop Sci 43: 350–357.

Jung, G., D.P. Coyne, J.M. Bokosi, J.R. Steadman & J. Nienhuis, 1998. Mapping genes for specific and adult plant resistance to rust and abaxial leaf pubescence and their genetic relationship using Random Amplified Polymorphic DNA (RAPD) markers in common bean. J Am Soc Hort Sci 123: 859–863.

Jung, G., D.P. Coyne, P. Skroch, J. Nienhuis, E. Arnaud-Santana, J. Bokosi, H. Ariyarathne, J. Steadman, J. Beaver & S. Kaeppler, 1996. Molecular markers associated with plant architecture and resistance to common blight, web blight, and rust in common beans. J Am Soc Hort Sci 121: 794–803.

Jung, G., P. Skroch, D.P. Coyne, J. Nienhuis, H. Ariyarathne, S. Kaeppler & M. Bassett, 1997. Molecular-marker-based genetic analysis of tepary-bean-derived common bacterial blight resistance in different developmental stages of common bean. J Am Soc Hort Sci 122: 329–337.

Jung, G., P.W. Skroch, J. Nienhuis, D.P. Coyne, E. Arnaud-Santana, H.M. Ariyarathne & J.M. Marita, 1999. Confirmation of QTL associated with common bacterial blight resistance in four different genetic backgrounds in common bean. Crop Sci 39: 1448–1455.

Kelly, J.D., 1997. A review of varietal response to bean common potyvirus in *Phaseolus vulgaris*. Plant Varieties Seeds 10: 1–6.

Kelly, J.D., L. Afanador & S.D. Haley, 1995. Pyramiding genes for resistance to bean common mosaic virus. Euphytica 82: 207–212.

Kelly, J.D., P. Gepts, P.N. Miklas & D.P. Coyne, 2003. Tagging and mapping of genes and QTL and molecular-marker assisted selection for traits of economic importance in bean and cowpea. Field Crops Res 82: 135–154.

Kelly, J.D., G.L. Hosfield, G.V. Varner, M.A. Uebersax, S.D. Haley & J. Taylor, 1994. Registration of 'Raven' black bean. Crop Sci 34: 1406–1407.

Kelly, J.D. & P.N. Miklas, 1998. The role of RAPD markers in breeding for disease resistance in common bean. Mol Breed 4: 1–11.

Kelly, J.D. & P.N. Miklas, 1999. Marker-assisted selection. In: S. Singh (Ed.), Common Bean Improvement in the Twenty-First Century, p. 93–123. Kluwer Academic Publishers, The Netherlands.

Kelly, J.D. & V.A. Vallejo, 2004. A comprehensive review of the major genes conditioning resistance to anthracnose in common bean. Hortscience 39:1196–1207.

Kipe-Nolt, J.A., H. Vargas & K.E. Giller, 1993. Nitrogen fixation in breeding lines of *Phaseolus vulgaris* L. Plant Soil 152: 103–106.

Kolkman, J.M. & J.D. Kelly, 2002. Agronomic traits affecting resistance to white mold in common bean. Crop Sci 42: 693–699.

Kolkman, J.M. & J.D. Kelly, 2003. QTL conferring resistance and avoidance to white mold in common bean. Crop Sci 43: 539–548.

Kornegay, J. & C. Cardona, 1991. Breeding for insect resistance in beans. In: A. Schoonhoven & O. van and Voysest (Eds.), Common Beans: Research for Crop Improvement, pp. 619–648.

Kornegay, J., C. Cardona & C.E. Posso, 1993. Inheritance of resistance to Mexican bean weevil in common bean, determined by bioassay and biochemical tests. Crop Sci 33: 589–594.

Kornegay, J., C. Cardona, J. van Esch & M. Alvarado, 1989. Identification of common bean lines with ovipositional resistance to *Empoasca kraemeri* (Homoptera: Cicadellidae). J Econ Entomol 82: 649–654.

Kornegay, J. & S.R. Temple, 1986. Inheritance and combining ability of leafhopper defense mechanisms in common bean. Crop Sci 26: 1153–1158.

Kyle, M.M. & R. Provvidenti, 1993. Inheritance of resistance to potyviruses in *Phaseolus vulgaris* L. II. Linkage relations and utility of a dominant gene for lethal systemic necrosis to soybean mosaic virus. Theor Appl Genet 86: 189–196.

Lamppa, R.S., P.L. Gross & L.E. del Rio, 2002. Races of *Pseudomonas syringae* pv. *phaeolicola* in North Dakota. Annu Rep Bean Improv Coop 45: 104–105.

Larsen, R.C. & P.N. Miklas, 2004. Generation and molecular mapping of a SCAR marker linked with the *Bct* gene for resistance to *beet curly top virus* in common bean. Phytopathology 94: 320–325.

Larsen, R.C., P.N. Miklas, K.L. Druffel & S.D. Wyatt, 2005. NL-3K isolate is a stable and naturally occurring interspecific recombinant derived from *Bean common mosaic necrosis virus* and *Bean common mosaic virus*. Phytopathology (in press).

Liao, H., X. Yan, G. Rubio, S.E. Beebe, M.W. Blair & J.P. Lynch, 2004. Basal root gravitropism and phosphorus acquisition efficiency in common bean. Funct Plant Biol 31: 1–12.

López, C.E., I.F. Acosta, C. Jara, F. Pedraza, E. Gaitán-Solís, G. Gallego, S. Beebe & J. Tohme, 2003. Identifying resistance gene analogs associated with resistances to different pathogens in common bean. Phytopathology 93: 88–95.

Lynch, J.P. & S.E. Beebe, 1995. Adaptation of beans (*Phaseolus vulgaris* L.) to low phosphorus availability. Hortscience. 30: 1165–1171.

Mahuku, G., C. Jara, C. Cajiao & S. Beebe, 2003. Sources of resistance to angular leaf spot (*Phaeoisariopsis griseola*) in common bean core collection, wild *Phaseolus vulgaris* and secondary gene pool. Euphytica 130: 303–313.

Mahuku, G., C. Jara, J.B. Cuasquer & G. Castellanos, 2002. Genetic variability within *Phaeoisariopsis griseola* from Central America and its implications for resistance breeding of common bean. Plant Pathology 51: 594–604.

Mahuku, G., C. Montoya, M.A. Henríquez, C. Jara, H. Teran & S. Beebe, 2004. Inheritance and characterization of angular leaf spot resistance gene present in common bean accession G 10474

and identification of an AFLP marker linked to the resistance gene. Crop Sci 44: 1817–1824.

Mayek-Pérez, N., C. López-Castaneda, E. López-Salinas & J.A. Acosta-Gallegos, 2001. Inheritance of genetic resistence to *Macrophomina phaseolina* (Tassi) Goid in common bean. Agrociencia 35: 637–648.

McKern, N.M., G.I. Mink, O.W. Barnett, A. Mishra, L.A. Whittaker, M.J. Silbernagel, C.W. Ward & D.D. Shukla, 1992. Isolates of bean common mosaic virus comprising two distinct potyviruses. Phytopathology 82: 923–928.

Melotto, M., L. Afanador & J.D. Kelly, 1996. Development of a SCAR marker linked to the *I* gene in common bean. Genome 39: 1216–1219.

Melotto, M., R.S. Balardin & J.D. Kelly, 2000. Host–pathogen interaction and variability of *Colletotrichum lindemuthianum*. In: D. Prusky, S. Freeman & M.B. Dickman (Eds.), *Colletotrichum* Host Specificity, Pathology, and Host–Pathogen Interaction, pp. 346–361. APS Press, St. Paul, MN.

Méndez-Vigo, B., C. Rodríguez-Suárez, A. Pañeda, J.J. Ferreira & R. Giraldez, 2005. Molecular markers and allelic relationships of anthracnose resistance gene cluster B4 in common bean. Euphytica 141: 237–245.

Mienie, C.M.S., M.M. Liebenberg, Z.A. Pretorius & P.N. Miklas, 2005. SCAR markers linked to the *Phaseolus vulgaris* rust resistance gene *Ur-13*. Theor Appl Genet (in press).

Miklas, P.N., 2005. List of DNA SCAR markers linked with disease resistance traits in bean. http://www.usda.prosser.wsu.edu/miklas/Scartable3.pdf.

Miklas, P.N., L. Afanador & J.D. Kelly, 1996a. Recombination-facilitated RAPD marker-assisted selection for disease resistance in common bean. Crop Sci 36: 86–90.

Miklas, P.N. & K.M. Bosak, 2004. Marker-assisted backcrossing of QTL for resistance to Sclerotinia white mold in pinto bean. Agron Abstr, Madison, WI.

Miklas, P.N., D.P. Coyne, K.F. Grafton, N. Mutlu, J. Reiser, D.T. Lindgren & S.P. Singh, 2003a. A major QTL for common bacterial blight resistance derives from the common bean great northern landrace cultivar Montana No. 5. Euphytica 131: 137–146.

Miklas, P.N., R. Delorme & R.H. Riley, 2003b. Identification of QTL conditioning resistance to white mold in a snap bean population. J Am Soc Hort Sci 128: 564–570.

Miklas, P.N., K.F. Grafton, D. Hauf & J.D. Kelly, 2005a. Registration of pinto bean germplasm lines USPT-WM-1 and USPT-WM-2 with partial resistance to white mold. Annu Rep Bean Improv Coop 48: 190–191.

Miklas, P.N., K.F. Grafton, J.D. Kelly, J.R. Steadman & M.J. Silbernagel, 1998a. Registration of four white mold resistant dry bean germplasm lines: I9365-3, I9365-5, I9365-31, and 92BG-7. Crop Sci 38: 1728.

Miklas, P.N., A.N. Hang, J.D. Kelly, C.A. Strausbaugh & R.L. Forster, 2002a. Registration of three kidney bean germplasm lines resistant to bean common mosaic virus and necrosis potyviruses: USLK-2 light red kidney, USDK-4 dark red kidney, and USWK-6 white kidney. Crop Sci 42: 674–675.

Miklas, P.N., D. Hauf, R. Henson & K.F. Grafton, 2004. Inheritance of ICA Bunsi-derived resistance in a navy × pinto bean cross. Crop Sci. 44: 1584–1588.

Miklas, P.N., E. Johnson, V. Stone, J.S. Beaver, C. Montoya & M. Zapata, 1996b. Selective mapping of QTL conditioning disease resistance in common bean. Crop Sci 36: 1344–1351.

Miklas, P.N., W.C. Johnson, R. Delorme, R.H. Riley & P. Gepts, 2001. Inheritance and QTL analysis of physiological resistance to white mold in common bean G122. Crop Sci 41: 309–315.

Miklas, P.N. & J.D. Kelly, 2002. Registration of two cranberry bean germplasm lines resistant to bean common mosaic and necrosis potyviruses: USCR-7 and USCR-9. Crop Sci 42: 673–674.

Miklas, P.N., J.D. Kelly, & S.P. Singh, 2003c. Registration of anthracnose-resistant pinto bean germplasm line USPT-ANT-1. Crop Sci 43: 1889–1890.

Miklas, P.N., R.C. Larsen, R. Riley & J.D. Kelly, 2000a. Potential marker-assisted selection for *bc-1*^{2} gene for resistance to bean common mosaic potyvirus in common bean. Euphytica 116: 211–219.

Miklas, P.N., M.A. Pastor-Corrales, G. Jung, D.P. Coyne, J.D. Kelly, P.E. McClean & P. Gepts, 2002b. Comprehensive linkage map of bean rust resistance genes. Annu Rep Bean Improv Coop 45: 125–129.

Miklas, P.N., J.R. Smith, R. Riley, K.F. Grafton, S.P. Singh, G. Jung & D.P. Coyne, 2000b. Marker-assisted breeding for pyramided resistance to common bacterial blight in common bean. Annu Rep Bean Improv Coop 43: 39–40.

Miklas, P.N., J.R. Smith & S.P. Singh, 2005b. Release of USDK-CBB-15 dark red kidney bean germplasm line with improved resistance to common bacterial blight. Annu Rep Bean Improv Coop 48: 192–193.

Miklas, P.N., J.R. Stavely & J.D. Kelly, 1993. Identification and potential use of a molecular marker for rust resistance in common bean. Theor Appl Genet 85: 745–749.

Miklas, P.N., V. Stone, M.J. Daly, J.R. Stavely, J.R. Steadman, M.J. Bassett, R. Delorme & J.S. Beaver, 2000c. Bacterial, fungal, and viral disease resistance loci mapped in a recombinant inbred common bean population ('Dorado'/XAN 176). J Am Soc Hort Sci 125: 476–481.

Miklas, P.N., V. Stone, C.A. Urrea, E. Johnson & J.S. Beaver, 1998b. Inheritance and QTL analysis of field resistance to ashy stem blight. Crop Sci 38: 916–921.

Mkandawire, A.B.C., R.B. Mabagala, P. Guzman, P. Gepts & R.L. Gilbertson, 2004. Genetic diversity and pathogenic variation of common blight bacteria (*Xanthomonas campestris* pv. *phaseoli* and *X. campestris* pv. *phaseoli* var. *fuscans*) suggests coevolution with the common bean. Phytopathology 94: 593–603.

Mmbaga, M., J.R. Steadman & J.R. Stavely, 1996. The use of host resistance in disease management of rust in common bean. Integrated Pest Manag Rev 1: 191–200.

Mukeshimana, G., A. Pañeda, C. Rodriguez, J.J. Ferreira, R. Giraldez & J.D. Kelly, 2005. Markers linked to the *bc-3* gene conditioning resistance to bean common mosaic potyviruses in common bean. Euphytica 144: 291–299.

Murray, J.D., T.E. Michaels, C. Cardona, A.W. Schaafsma & K.P. Pauls, 2004a. Quantitative trait loci for leafhopper (*Empoasca fabae* and *Empoasca kraemeri*) resistance and seed weight in the common bean. Plant Breed 123: 474–479.

Murray, J.D., T.E. Michaels, K.P. Pauls, C. Cardona & A.W. Schaafsma, 2004b. Yield and insect injury in leafhopper (*Empoasca fabae* Harris and *Empoasca kraemeri* Ross & Moore) infested dry beans in Ontario and Colombia. Can J Plant Sci 84: 891–900.

Murray, J.D., T.E. Michaels, K.P. Pauls & A.W. Schaafsma, 2001. Determination of traits associated with leafhopper (*Empoasca fabae* and *Empoasca kraemeri*) resistance and dissection of leafhopper damage symptoms in the common bean (*Phaseolus vulgaris*). Ann Appl Biol 139: 319–327.

Mutlu, N., P.N. Miklas, J. Reiser & D.P. Coyne, 2005a. Backcross breeding for improved resistance to common bacterial blight in pinto bean (*Phaseolus vulgaris* L.). Plant Breed 124: 282–287.

Mutlu, N., P.N. Miklas, J.R. Steadman, A.V. Vidaver, D. Lindgren, J. Reiser & M.A. Pastor-Corrales, 2005b. Registration of pinto bean germplasm line ABCP-8 with resistance to common bacterial blight. Crop Sci 45: 806.

Navarrete-Maya, R.E. Trejo-Albarran, J. Navarrete-Maya, J.M. Prudencio Sains & J.A. Acosta-Gallegos, 2002. Reaction of bean genotypes to *Fusarium* sp. and *Rhizoctonia solani* in central Mexico. Annu Rep Bean Improv Coop 45: 154–155.

Navarro, F., M. Sass & J. Nienhuis, 2003. Identification and mapping bean root rot resistance in a population of Mesoamerican × Andean origin. Annu Rep Bean Improv Coop 46: 213–214.

Nietsche, S., A. Borem, G.A. Carvalho, R.C. Rocha, T.J. Paula, E.G. Barros & M.A. Moreira, 2000. RAPD and SCAR markers linked to a gene conferring resistance to angular leaf spot in common bean. J Phytopathol 148: 117–121.

Nodari, R.O., S.M. Tsai, P. Guzman, R.L. Gilbertson & P. Gepts, 1993. Towards an integrated linkage map of common bean. III. Mapping genetic factors controlling host–bacteria interactions. Genetics 134: 341–350.

Olaya, G., G.S. Abawi & N.F. Weeden, 1996. Inheritance of the resistance to *Macrophomina phaseolina* and identification of RAPD markers linked to the resistance genes in beans. Phytopathology 86: 674–679.

Oliveira, E.J., A.L. Alzate-Marin, C.L.P. Melo, A. Borém, E.G. Barros & M.A. Moreira, 2002. Backcross assisted by RAPD markers for the introgression of angular leaf spot resistance genes in common bean cultivars. Annu Rep Bean Improv Coop 45: 142–143.

Osborn, T.C., T. Blake, P. Gepts & F. Bliss, 1986. Bean Arcelin 2. Genetic variation, inheritance and linkage relationships of a novel seed protein of *Phaseolus vulgaris* L. Theor Appl Genet 71: 847–855.

Osborn, T.C., D.C. Alexander, S. Sun, C. Cardona & F. Bliss, 1988. Insecticidal activity and lectin homology of arcelin seed protein. Science 240: 207–210.

Osorno, J.M., J.S. Beaver, F.H. Ferwerda & P.N. Miklas, 2003. Two genes from *Phaseolus coccineus* L. confer resistance to bean golden yellow mosaic virus. Annu Rep Bean Improv Coop 46: 147–148.

Park, S.O., D.P. Coyne, J.R. Steadman, K.M. Crosby & M.A. Brick, 2004. RAPD and SCAR markers linked to the *Ur-6* Andean gene controlling specific rust resistance in common bean. Crop Sci 44: 1799–1807.

Park, S.O., D.P. Coyne, J.R. Steadman & P.W. Skroch, 2001. Mapping of QTL for resistance to white mold diseases in common bean. Crop Sci 41: 1253–1262.

Park, S.O., D.P. Coyne, J.R. Steadman & P.W. Skroch, 2003. Mapping of the *Ur-7* gene for specific resistance to rust in common bean. Crop Sci 43: 1470–1476.

Pastor-Corrales, M.A., 2003. Sources, genes for resistance, and pedigrees of 52 rust and mosaic resistant dry bean germplasm lines released by the USDA Beltsville Bean Project in collaboration with Michigan, Nebraska, and North Dakota Agricultural Experiment Stations. Annu Rpt Bean Improv Coop 46: 235–241.

Pastor-Corrales, M.A. & C. Jara, 1995. La evolución de *Phaeoisariopsis griseola* con el frijol común en América Latina. Fitopatol Colomb 19: 15–24.

Pastor-Corrales, M.A., C. Jara & S.P. Singh, 1998. Pathogenic variation in, sources of, and breeding for resistance to *Phaeoisariopsis griseola* causing angular leaf spot in common bean. Euphytica 103: 161–171.

Patterson, A.H., 1995. Molecular dissection of quantitative traits: Progress and prospects. Genome Res 5: 321–333.

Petzoldt, R. & M.H. Dickson, 1996. Straw test for resistance to white mold in beans. Annu Rep Bean Improv Coop 39: 142–143.

Pillemer, E.A. & W.M. Tingey, 1976. Hooked trichomes: A physical barrier to a major agricultural pest. Science 193: 482–474.

Pyndji, M.M. & R. Trutmann, 1992. Managing angular leaf spot on common bean in Africa by supplementing farmer mixtures with resistant varieties. Plant Dis 76: 1144–1147.

Queiroz, V.T., C.S. Sousa, M.R. Costa, D.A. Sanglad, K.M.A. Arruda, T.L.P.O. Souza, V.A. Ragagnin, E.G. Barros & M.A. Moreira, 2004. Development of SCAR markers linked to common bean angular leaf spot resistance genes. Annu Rep Bean Improv Coop 47: 237–238.

Ragagnin, V.A., D.A. Sanglard, T.L.P.O. de Souza, M.A. Moreira & E.G. de Barros, 2003. Simultaneous transfer of resistance genes for rust, anthracnose, and angular leaf spot to cultivar Perola assisted by molecular markers. Annu Rep Bean Improv Coop 46: 159–160.

Ramirez-Vallejo, P. & J.D. Kelly, 1998. Traits related to drought resistance in common bean. Euphytica 99: 127–136.

Rao, I.M., 2001. Role of physiology in improving crop adaptation to abiotic stresses in the tropics: The case of common bean and tropical forages. In: M. Pessarakli (Ed.), Handbook of Plant and Crop Physiology, pp. 583–613. Marcel Dekker, New York, USA.

Rivkin, M.I., C.E. Vallejos & P.E. McClean, 1999. Disease-resistance related sequences in common bean. Genome 42: 41–47.

Román-Avilés, B. & J.D. Kelly, 2005. Identification of quantitative trait loci conditioning resistance to *Fusarium* root rot in common beans. Crop Sci 45: in press.

Sandlin, C.M., J.R. Steadman, C.M. Araya & D.P. Coyne, 1999. Isolates of *Uromyces appendiculatus* with specific virulence to landraces of *Phaseolus vulgaris* of Andean origin. Plant Dis 83: 108–113.

Santos, A.S., R.E. Bressan-Smith, M.G. Pereira, R. Rodrigues & C.F. Ferreira, 2003. Genetic linkage map of *Phaseolus vulgaris* and indentification of QTLs responsible for resistance to *Xanthomonas axonopodis* pv. *phaseoli*. Fitopatol Bras 28: 5–10.

Santos-Filho, H.P., S. Ferraz & C. Vieira, 1976. Resisténcia à mancha-angular (*Isariopsis griseola* Sacc.) no feijoeiro comum (*Phaseolus vulgaris* L.). Rev Ceres 23: 226–230.

Sartorato, A., S. Nietsche, E.G. Barros & M.A. Moreira, 2000. RAPD and SCAR markers linked to resistance gene to angular leaf spot in common beans. Fitopatol Bras 25: 637–642.

Schneider, K.A., M.E. Brothers & J.D. Kelly, 1997a. Marker-assisted selection to improve drought tolerance in common bean. Crop Sci. 37: 51–60.

Schneider, K.A., K.F. Grafton & J.D. Kelly, 2001. QTL analysis of resistance to *Fusarium* root rot in bean. Crop Sci 41: 535–542.

Schneider, K.A., R. Rosales-Serna, F. Ibarra-Pérez, B. Cazares-Enriquez, J.A. Acosta-Gallegos, P. Ramirez-Vallejo, N. Wassimi & J.D. Kelly, 1997b. Improving common bean performance under drought stress. Crop Sci 37: 43–50.

Schoonhoven, A., C. Cardona & J. Valor, 1983. Resistance to the bean weevil and the Mexican bean weevil (Coleoptera:

Bruchidae) in noncultivated common bean accessions. J Econ Entomol 76: 1255–1259.

Schultz, H.K. & L.L. Dean, 1947. Inheritance of curly top disease reaction in the bean, *Phaseolus vulgaris*. J Am Soc Agron 39: 47–51.

Schwartz, H.F., D.H. Casciano, J.A. Asenga & D.R. Wood, 1987. Field measurement of white mold effects upon dry beans with genetic resistance or upright plant architecture. Crop Sci 27: 699–702.

Schwartz, H.F., K. Otto, H. Terán & S.P. Singh, 2004. Inheritance of white mold resistance in the interspecific crosses of pinto bean cultivars Othello and *Phaeolus coccineus* L. accessions PI 433246 and PI 439534. Annu Rep Bean Imorov Coop 47: 279–280.

Schwartz, H.F., M.A. Pastor-Corrales & S.P. Singh, 1982. New sources of resistance to anthracnose and angular leaf spot of beans (*Phaseolus vulgaris* L.). Euphytica 31: 741–754.

Silbernagel, M.J. & R.M. Hannan, 1992. Use of plant introductions to develop U.S. bean cultivars. In: H. Shands & L.E. Weisner (Eds.), Use of Plant Introductions in Cultivar Development. Part 2. CSSA Special Publication 20, pp. 1–8. CSSA, Madison, WI.

Singh, S.P., 1995. Selection for water stress tolerance interracial populations of common bean. Crop Sci 35: 118–124.

Singh, S.P., C. Cardona, F.J. Morales, M.A. Pastor-Corrales & O. Voysest, 1998. Gamete selection for upright carioca bean with resistance to five diseases and a leafhopper. Crop Sci 38: 666–672.

Singh, S.P., P. Gepts & D.G. Debouck, 1991. Races of common bean (*Phaseolus vulgaris*, Fabaceae). Econ Bot 45: 379–396.

Singh, S.P., F.J. Morales, P.N. Miklas & H. Terán, 2000a. Selection for bean golden mosaic resistance in intra- and inter-racial bean populations. Crop Sci 40: 1565–1572.

Singh, S.P., F.J. Morales & H. Terán, 2000b. Registration of bean golden mosaic resistant dry bean germplasm GMR 1 and GMR 5. Crop Sci 40: 1836.

Singh, S.P. & C.G. Muñoz, 1999. Resistance to common bacterial blight among *Phaseolus* species and common bean improvement. Crop Sci 39: 80–89.

Singh, S.P., C.G. Muñoz & H. Terán, 2001. Registration of common bacterial blight resistant dry bean germplasm VAX 1, VAX 3, and VAX 4. Crop Sci 41: 275–276.

Singh, S.P., H. Terán, J.A. Gutiérrez, M.A. Pastor-Corrales, H.F. Schwartz & F.J. Morales, 2003. Registration of A 339, MAR 1, MAR 2, and MAR 3 angular-leaf-spot and anthracnose-resistant germplasm. Crop Sci 43: 1886–1887.

Snapp, S., W. Kirk, B. Román-Avilés & J. Kelly, 2003. Root traits play a role in integrated management of Fusarium root rot in snap beans. Hortscience 38: 187–191.

Snoeck, C., J. Vanderleyden & S. Beebe, 2003. Strategies for genetic improvement of common bean and rhizobia towards efficient interactions. Plant Breed Rev 23: 21–72.

Sparvoli, F. & R. Bollini, 1998. Arcelin in wild bean (*Phaseolus vulgaris* L.) seeds: Sequence of arcelin 6 shows it is a member of the arcelin 1 and 2 subfamily. Gen Resources Crop Evol 45: 383–388.

Specht, J.E., D.J. Hume & S.V. Kumudini, 1999. Soybean yield potential – A genetic and physiological perspective. Crop Sci 39: 1560–1570.

Spence, N.J. & D.G.A. Walkey, 1995. Variation for pathogenicity among isolates of bean common mosaic virus in Africa and reinterpretation of the genetic relationship between cultivars of *Phaseolus vulgaris* and pathotypes of BCMV. Plant Pathol 44: 527–546.

Sponchiado, B.N., J.W. White, J.A. Castillo & P.G. Jones, 1989. Root growth of four common bean cultivars in relation to drought tolerance in environments with contrasting soil types. Exp Agric 25: 249–257.

Stavely, J.R., 1984. Genetics of resistance to *Uromyces phaseoli* in a *Phaseolus vulgaris* line resistant to most races of the pathogen. Phytopathology 74: 339–344.

Stavely, J.R., J.D. Kelly, K.F. Grafton, C.A. Mullins, A. Straw, R.T. McMillan Jr., J.S. Beaver, P.N. Miklas, J. Steinke, J.R. Steadman, D.P. Coyne, D.T. Lindgren & M.J. Silbernagel, 1997. Rust resistant bean germplasm releases, 1994–1996. Annu Rep Bean Improv Coop 40: 120–121.

Stavely, J.R., R.T. McMillan, J.S. Beaver & P.N. Miklas, 2001. Release of three McCaslan type, indeterminate, rust and golden mosaic resistant snap bean germplasm lines BelDade RGMR 4, 5 and 6. Annu Rep Bean Improv Coop 44: 197–198.

Stenger, D.C., G.N. Revington, M.C. Stevenson & D.M. Bisaro, 1991. Replicational release of geminivirus genomes from tandemly repeated copies: Evidence for rolling-circle replication of a plant viral DNA. Proc Natl Acad Sci USA 88: 8029–8033.

Strausbaugh, C.A., J.R. Myers, R.L. Forster & P.E. McClean, 1999. *Bc-1* and *Bc-u* two loci controlling bean common mosaic virus resistance in common bean are linked. J Am Soc Hort Sci 124: 644–648.

Sutton, L.A. & D.P. Coyne, 2002. Vegetable cultivar descriptions for North America: Bean-dry, lists 1–26 combined. Hortscience, http://cuke.hort.ncsu.edu/wehner/vegcult/beandry.html.

Tar'an, B., T.E. Michaels & K.P. Pauls, 2001. Mapping genetic factors affecting the reaction to *Xanthomonas axonopodis* pv. *phaseoli* in *Phaseolus vulgaris* L. under field conditions. Genome 44: 1046–1056.

Taylor, J.D., D.M. Teverson, M.A. Allen & M.A. Pastor-Corrales, 1996a. Identification and origin of races of *Pseudomonas syringae* pv. *phaseolicola* from Africa and other bean growing areas. Plant Pathol 45: 469–478.

Taylor, J.D., D.M. Teverson & J.H.C. Davis, 1996b. Sources of resistance to *Pseudomonas syringae* pv. *phaseolicola* races in *Phaseolus vulgaris*. Plant Pathol 45: 479–485.

Terán, H. & S.P. Singh, 2002. Comparison of sources and lines selected for drought resistance in common bean. Crop Sci 42: 64–70.

Thomas, C.V. & J.G. Waines, 1984. Fertile backcross and allotetraploid plants from crosses between tepary beans and common beans. J Hered 75: 93–98.

Toubart, P., A. Desiderio, G. Salvi, F. Cervone, L. Daroda & G. De Lorenzo, 1992. Cloning and characterization of the gene encoding the endopolygalacturonase-inhibiting protein (Pgip) of *Phaseolus vulgaris* L. Plant J 2: 367–373.

Tsai, S.M., R.O. Nodari, D.H. Moon, L.E.A. Camargo, R. Vencovsky & P. Gepts, 1998. QTL mapping for nodule number and common bacterial blight in *Phaseolus vulgaris* L. Plant Soil 204: 135–145.

Urrea, C.A., P.N. Miklas, J.S. Beaver & R.H. Riley, 1996. A codominant randomly amplified polymorphic DNA (RAPD) marker useful for indirect selection of BGMV resistance in common bean. J Am Soc Hort Sci 121: 1035–1039.

Vadez, V., J.H. Lasso, D.P. Beck & J.J. Drevon, 1999. Variability of N_2-fixation in common bean (*Phaseolus vulgaris* L.) under P deficiency is related to P use efficiency: N_2-fixation tolerant to P deficiency. Euphytica 106: 231–242.

Vallad, G., M. Rivkin, C. Vallejos & P. McClean, 2001. Cloning and homology modelling of a Pto-like protein kinase family of common bean (*Phaseolus vulgaris* L.). Theor Appl Genet 103: 1046–1058.

Vallejos, C.E., J.J. Malandro, K. Sheehy & M.J. Zimmermann, 2000. Detection and cloning of expressed sequences linked to a target gene. Theor Appl Genet 101: 1109–1113.

VandenBosch, E. & G. Stacey, 2003. Summaries of legume genomics projects from around the globe. Community resources for crops and models. Plant Physiol 131: 840–865.

Vandemark, G.J. & P.N. Miklas, 2002. A fluorescent PCR assay for the codominant interpretation of a dominant SCAR marker linked to the virus resistance allele *bc-1*^{2} in common bean. Mol Breed 10: 193–201.

Vandemark, G.J. & P.N. Miklas, 2005. Genotyping common bean for the potyvirus resistance alleles I and *bc-1*^{2} with a multiplex real-time polymerase chain reaction assay. Phytopathology 95: 499–505.

Velez, J.J., M.J. Bassett, J.S. Beaver & A. Molina, 1998. Inheritance of resistance to bean golden mosaic virus in common bean. J Am Soc Hort Sci 123: 628–631.

Wallace, D.H. & R.E. Wilkinson, 1975. Breeding for resistance in dicotyledonous plants to root rot fungi. In: G.W. Bruehll (Ed.), Biology and Control of Soil-Borne Plant Pathogens. American Phytopathological Society, St. Paul, MN.

Walter, M.H., J. Liu, C. Grand, C.J. Lamb & D. Hess, 1990. Bean-pathogenesis-related proteins deduced from elicitor-induced transcripts are members of a ubiquitous new class of conserved PR proteins including pollen allergens. Mol Gen Genet 222: 353–360.

Webb, K.J., L. Skot, S. Mizen, M. Paniske, B. Jorgensen, M. Nicholson, A. Altman, M. Ziv & S. Izhar, 1999. Identification of genes expressed in roots and nodules of *Lotus japonicus*. In: Plant Biotechnology and In: Vitro Biology in the 21st Century, Vol. 36, pp. 227–229. Proceedings of the IXth International Congress of the International Association of Plant Tissue Culture and Biotechnology, Jerusalem, Israel, 14–19 June 1998. Current Plant Science and Biotechnology in Agriculture.

White, J.W., 1987. Preliminary results of the Bean International Drought Yield Trial (BIDYT). In: Proceedings of the International Bean Drought Workshop, 19–21 October 1987, p. 126–145. Cali, Colombia.

White, J.W., R. Ochoa, F. Ibarra & S.P. Singh, 1994. Inheritance of seed yield, maturity and seed weight of common bean (*Phaseolus vulgaris* L.) under semi-arid rainfed conditions. J Agric Sci 122: 265–273.

Wortmann, C.S., R.A. Kirkby, C.A. Eledu & D.J. Allen, 1998. Atlas of common bean (*Phaseolus vulgaris* L.) production in Africa. CIAT, Cali, Colombia.

Yan, X., S.E. Beebe & J.P. Lynch, 1995a. Genetic variation for phosphorus efficiency of common bean in contrasting soil types. II. Yield response. Crop Sci 35: 1094–1099.

Yan, X., H. Liao, S.E. Beebe, M.W. Blair & J.P. Lynch, 2004. Molecular mapping of QTLs associated with root hairs and acid exudation as related to phosphorus uptake in common bean. Plant and Soil 265: 17–29.

Yan, X., H. Liao, M.C. Trull, S.E. Beebe & J.P. Lynch, 2001. Induction of a major leaf acid phosphatase does not confer adaptation to low phosphorus availability in common bean. Plant Phys 125: 1–11.

Yan, X., J.P. Lynch & S.E. Beebe, 1995b. Genetic variation for phosphorus efficiency of common bean in contrasting soil types. I. Vegetative response. Crop Sci 35: 1086–1093.

Yan, X., J.P. Lynch & S.E. Beebe, 1996. Utilization of phosphorus substrates contrasting common bean genotypes. Crop Sci 36: 936–941.

Yencho, G.C., M.B. Cohen & P.F. Byrne, 2000. Applications of tagging and mapping insect resistance loci in plants. Annu Rev Entomol 45: 393–422.

Young, R.A., M. Melotto, R.O. Nodari & J.D. Kelly, 1998. Marker assisted dissection of the oligogenic anthracnose resistance in common bean cultivar, G 2333. Theor Appl Genet 96: 87–94.

Yu, K., S.J. Park & V. Poysa, 2000. Marker-assisted selection of common beans for resistance to common bacterial blight: Efficiency and economics. Plant Breed 119: 411–415.

Yu, K., S.J. Park, B. Zhang, M. Haffner & V. Poysa, 2004. An SSR marker in the nitrate reductase gene of common bean is tightly linked to a major gene conferring resistance to common bacterial blight. Euphytica 138: 89–95.

Yu, Z.H., R.E. Stall & C.E. Vallejos, 1998. Detection of genes for resistance to common bacterial blight of beans. Crop Sci 38: 1290–1296.